A Dissertation on

PREVALENCE AND CLINICAL CHARACTERISTICS OF HYPERTENSIVE EMERGENCIES IN TERTIARY CARE TEACHING INSTITUTE, CHENNAI.

Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI – 600032

In partial fulfilment of the Regulations
for the Award of the Degree of

M.D. BRANCH - I

GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE
STANLEY MEDICAL COLLEGE
CHENNAI – 600 001
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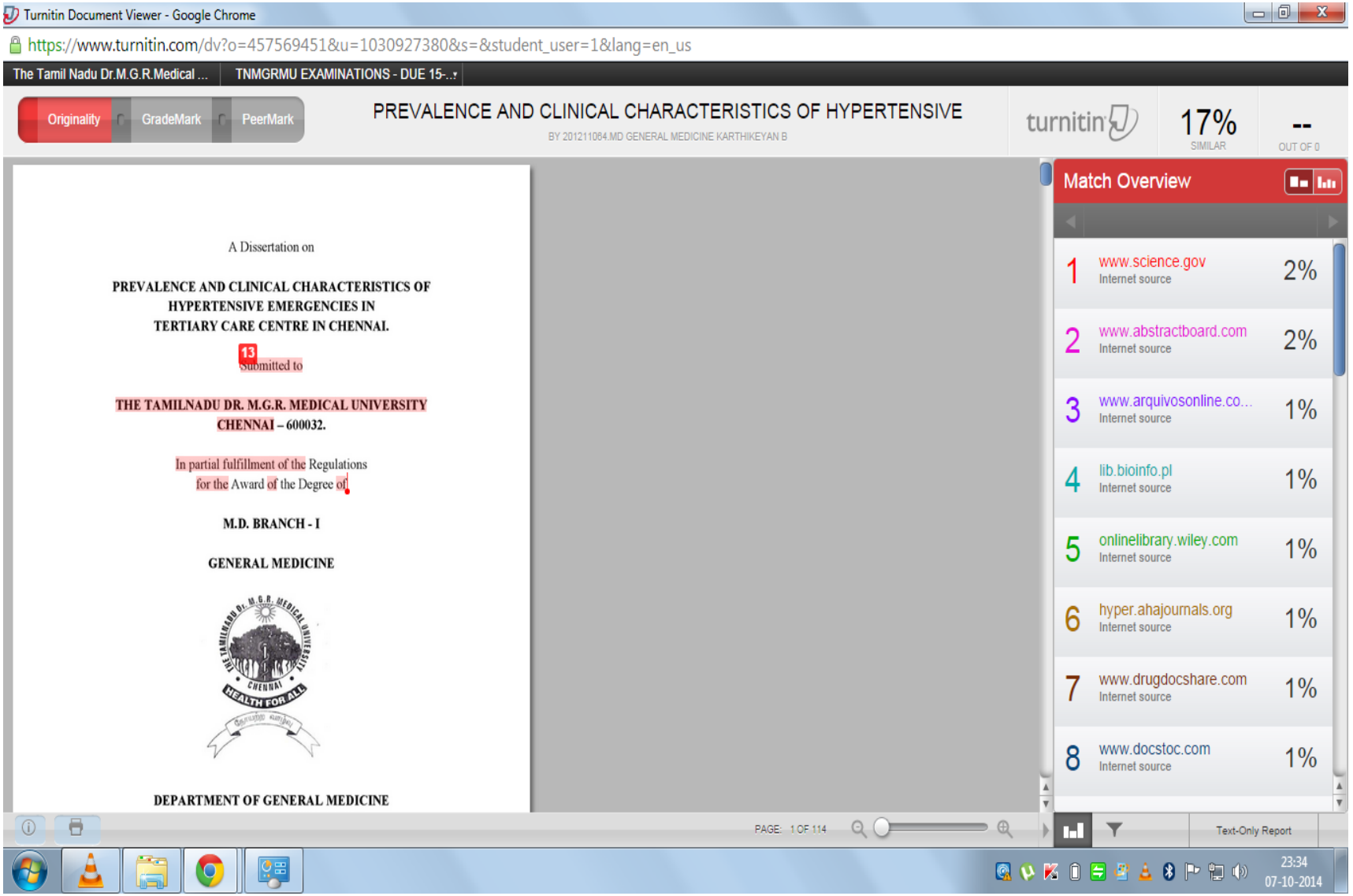
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This is to certify that **DR. B. KARTHIKEYAN,** Post - Graduate Student (MAY 2012 TO APRIL 2015) in the Department of General Medicine STANLEY MEDICAL COLLEGE, Chennai- 600 001, has done this dissertation on "**PREVALENCE AND CLINICAL CHARACTERISTICS OF HYPERTENSIVE EMERGENCIES IN TERTIARY CARE TEACHING INSTITUTE, CHENNAI – 600001"** under my guidance and supervision in partial fulfilment of the regulations laid down by the Tamil Nadu Dr. M.G.R. Medical University, Chennai, for M.D. (General Medicine), Degree Examination to be held in April 2015.

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DECLARATION

I **DR. B. KARTHIKEYAN** declare that I carried out this work on **"PREVALENCE AND CLINICAL CHARACTERISTICS OF HYPERTENSIVE EMERGENCIES IN TERTIARY CARE TEACHING INSTITUTE, CHENNAI - 600001**" at the Medical OPD, Cardiology OPD and Medical wards of Government Stanley Hospital during the period November 2013 to November 2014. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, or diploma to any other university, board either in India or abroad.

This is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the rules and regulation for the M. D. Degree examination in General Medicine.

Dr. B. KARTHIKEYAN

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TABLE OF CONTENTS

ABBREVIATIONS

SBP	SYTOLIC BLOOD PRESSURE
DBP	DIASTOLIC BLOOD PRESSURE
MAP	MEAN ARTERIAL PRESSURE
CHF	CONGESTIVE HEART FAILURE
AKI	ACUTE KIDNEY INJURY
CO	CARDIAC OUTPUT
HE	HYPERTENSIVE EMERGENCY
ICH	INTRACEREBRAL HAEMORRHAGE
SAH	SUB ARACHNOID HAEMORRHAGE
ACE	ANGIOTENSIN CONVERTING ENZYME
PIH	PREGNANCY INDUCED HYPERTENSION

Prevalence and clinical characteristics of hypertensive emergencies in tertiary care teaching institute, Chennai.

Abstract

Aims

To assess the prevalence of hypertensive emergency among the hospital admission over the period of 2 months and to assess the risk factors, clinical presentation and profile of patients.

Methods

It is a cross sectional descriptive hospital based study done in patients admitted in admitting unit and medicine ward of Stanley medical college. The patients more than 18 years of age and severe elevations in BP (>180/120 mmHg) associated with evidence of progressive target organ damage at the time of admission (JNC 7 criteria) were included in the study. On admission detailed history, examination and relevant investigations will be done.

Results

The study population contained 82 patients, 43 were male and 39 were female. 42 % were elderly; headache and giddiness were the most common presentation. One third of patients were newly diagnosed as having hypertension, among the causes 93 % were essential hypertension and the 7 %

were secondary hypertension all of them are having renal disease. 77 % were recently diagnosed hypertension less than 5 years and 48 % were on irregular treatment and follow up. The mean systolic BP is 202 and the diastolic BP is 127 mm Hg. The hypertensive retinopathy were present in 58% of patients and 8 % were having papilledema. ECG changes were present in 52 % of patients and 38% were having stroke. System wise the most common is neurological in which intracerebral haemorrhage is most common. The second most common presentation is cardiovascular consisting of acute left ventricular failure.

Conclusion

The prevalence of hypertensive emergencies in our setup is 0.68. In this 42 % were more than 60 years. Headache and giddiness were the most common presenting symptom. The hypertension was undiagnosed in nearly one third of patients. In hypertension 93 % were essential hypertension and 7% is due to renal failure. Nearly 50% of hypertensive emergencies were on irregular treatment and follow up. Majority were recently diagnosed. The most common system wise presentation is neurological. The second most common is cardiovascular system.

Introduction

Cardiovascular deaths are the leading cause of death globally, many people die from cardiovascular diseases than from any other disease, accounting nearly 30% of all global deaths. The number of people who will die due to cardiovascular disease is expected to increase by 2030. Cardiovascular diseases are expected to remain the single most important cause of death (1).

Hypertension is one of the important factors attributing for cardiovascular disease mortality and morbidity. Arterial hypertension affects approximately one billion people worldwide and in approximately 30% it is undiagnosed. It is noted that in India the population above 60 has increased drastically. The number of peoples older than 60 years is expected to increase in the next 3 decades. This will lead to a proportional increase in the prevalence of hypertension in the India. The prevalence of hypertension in India is between 20–40% in urban adults and 12–17% among rural adults (2).

Normal blood pressure is defined as a SBP of less than 120 mm Hg and DBP of less than 80 mm Hg. Hypertension is defined as a SBP of 140 mm Hg or higher or a DBP of 90 mm Hg or higher.

A SBP of 120 to 139 mm Hg or a DBP of 80 to 89 mm Hg is considered prehypertension according to JNC VII since people in this range of BP are observed to have higher tendency for hypertension over time. There is a

continuous relationship between hypertension level and cardiac risk. The maximum blood pressure and the duration of blood pressure mainly determine the outcome. Many patients who are chronically uncontrolled in blood pressure suffer end-organ damage over period of time (3).

The chronic hypertension is a well-known risk factor for cardiovascular, cerebrovascular and renal disease and the acute elevations in BP can lead to acute target organ damage with significant mortality and morbidity. Hypertensive crisis is commonly seen in the emergency department. The Prompt identification, evaluation and treatment of hypertensive crisis is crucial to prevent permanent target organ damage.

The patients with untreated and inadequately treated hypertensive are most commonly prone to acute elevation in the blood pressures.

The term hypertensive crisis includes two entities hypertensive emergencies and urgencies.

The hypertensive emergency is severe elevation in blood pressure with acute target organ damage which includes stroke, left ventricular failure, retinopathy and renal failure. Most of the target organ damage occurs when DBP exceeds 120 to 130 mm Hg. In HE, immediate reduction is essential to preventing the long term damage and this mostly requires intravenous parenteral drugs.

The significantly elevated BP but without any evidence of acute target organ damage is defined as hypertensive urgency. In these patients reduction in blood pressure can be achieved over a days mostly with oral drugs and usually do not require any intensive care monitoring (3).

Currently, with the improvement in the drugs available for the treatment of hypertension, the estimation of 1 to 2% of hypertensive can develop hypertensive emergency during their lifetime (7).

In spite of the development of new antihypertensive medications over the last 3-4 decades the incidence of HE has not come down and in fact it has increased.

There is inadequate study in India for prevalence of hypertensive crisis among emergency hospital admission, only fewer studies were available exploring the various presentation of hypertensive emergency. This study aims to bring out the possible data about hypertensive emergencies in hospital setup.

Review of literature

Hypertension is one of the leading causes of death worldwide. Globally cardiovascular disease accounts to 17 million deaths a year. In these, hypertension accounts for 9.4 million deaths in the world every year. Hypertension is responsible for 45% of deaths due to heart disease and 51% of deaths due to stroke.

Hypertension is the risk factor for

1. CAD
2. CHF
3. Cerebral infarction
4. Intra cerebral haemorrhage
5. Kidney failure
6. Peripheral vascular disease.

It mostly associated with other cardiovascular disease risk factors and the risk of cardiovascular disease increases with other risk factors. Worldwide large segments of the hypertensive are either not diagnosed or not adequately treated for hypertension.

Epidemiology of hypertension.

The prevalence of hypertension varies between countries and among populations within a country. Hypertension is prevalent in all populations except in few small populations living in isolated settings of environment. In developed countries, blood pressure increases during the first two decades of life and this is associated with maturation of the person and also the growth. Blood pressure observes tracking phenomenon in children and during adolescence. In elderly aged 60 and older, SBP of women are higher than that of men. In adults DBP increases with age up to 60 years, after that it decreases. The leads to widening in pulse pressure after age of 60. The probability of elderly person developing hypertension during his lifetime is nearly 90%.

The environmental factor and also the genetic factors attribute to regional variations of blood pressure and prevalence in hypertension. Studies of acculturation and migration population shows there is a profound environmental contribution to blood pressure.

Risk factors for hypertension

1. Obesity and weight gain
2. Dietary Na Cl intake.
3. Low calcium intake
4. Low potassium intake
5. Alcohol consumption
6. Psychosocial stress
7. Physical inactivity

The twin studies and the family studies indicate a heritable component in elevated blood pressure. Family studies indicate that blood pressure heritability is in the 15–35% range. Twin studies indicate heritability estimate of 60% for males and 40% for females.

Genetic Considerations

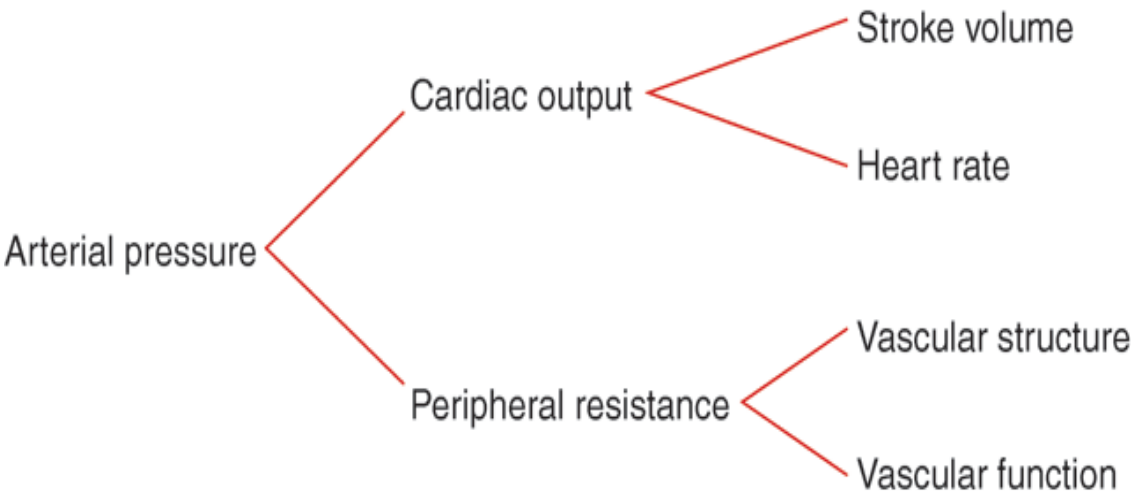
Hypertension is a polygenetic disorder in which genes and an environmental exposure contributes to blood pressure. The different sets of genes leads to various phenotypes associated with blood pressure,

Genes possibly associated with hypertension.

1. Gene encoding angiotensinogen
2. Angiotensin-converting enzyme
3. The alpha adducin gene – responsible for absorption of sodium.
4. Aldosterone synthase
5. Adrenoreceptor.

The evidence suggests that there may be genetic determinants of target organ damage attributable to hypertension. Family studies show significant heritability of left ventricular mass and there is significant individual variation in the responses of the heart to hypertension. Studies show that genetic factors also may contribute to hypertensive nephropathy. Specific genetic variants have been identified to coronary artery disease and stroke.

Mechanisms of Hypertension



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com

Cardiac output and the peripheral vascular resistance are the two important determinant factors of blood pressure. Cardiac output is mainly determined by heart rate and stroke volume. The stroke volume is determined by myocardial contraction capacity and also the size of the blood vascular compartment. Peripheral arterial resistance is determined by anatomical and functional changes in the small arteries and arterioles.

Intravascular Volume

Vascular volume is principle determining factor of blood pressure in long term. Sodium is an extracellular ion and it is a principle determinant of the ECF. When intake of sodium exceeds the limit of the kidneys to excrete sodium, vascular compartment expands and leading to increase in cardiac output. The vascular beds in brain and kidney auto regulate their blood flow and for constant blood flow the arterial pressure increases. During the course of time peripheral vascular resistance increases and the cardiac output become normal.

The effect of sodium on pressure is mainly related to the intake of sodium and chloride. A non-chloride salt of sodium has insignificant effect on blood pressure. To maintain arterial pressure urinary sodium excretion increases and the sodium balance is maintained because of pressure natriuresis phenomenon due to atrial natriuretic peptides. Larger increases in BP are required to achieve natriuresis in patients with decreased capacity to excrete sodium.

Na Cl dependent hypertension may be due to

1. Diminished capacity of kidney to excrete the sodium
2. Intrinsic renal disease
3. Increased production of mineralocorticoid
4. Increased sympathetic activity to the kidney.

Autonomic Nervous System

ANS controls cardiovascular homeostasis by

1. Pressure
2. Volume
3. Chemoreceptor signals.

The three endogenous catecholamines are

1. Noradrenaline
2. Adrenaline
3. Dopamine

The activities of these receptors are due to G protein coupled receptor and by downstream second messengers. The receptor affinity, density and physiologic responsiveness to catecholamines are the determining factors.

Circulating catecholamine concentrations determine the number of adrenoreceptors in many tissues. Down regulation of receptors occurs due to sustained high levels of catecholamines leading to tachyphylaxis, to catecholamines. Chronic administration of drugs that blocking adrenergic receptors results in upregulation of sensitivity to sympathetic stimuli.

Baroreflex mediated by sensory nerve endings in aorta and carotid sinus controls blood pressure in a minute to minute basis. When the pressure increases the rate of firing increases and it leads to decrease sympathetic outflow by decrease in arterial pressure and heart rate. The reverse occurs when the pressure decreases.

This is a principle mechanism for rapid control of fluctuations in arterial pressure that can occur during

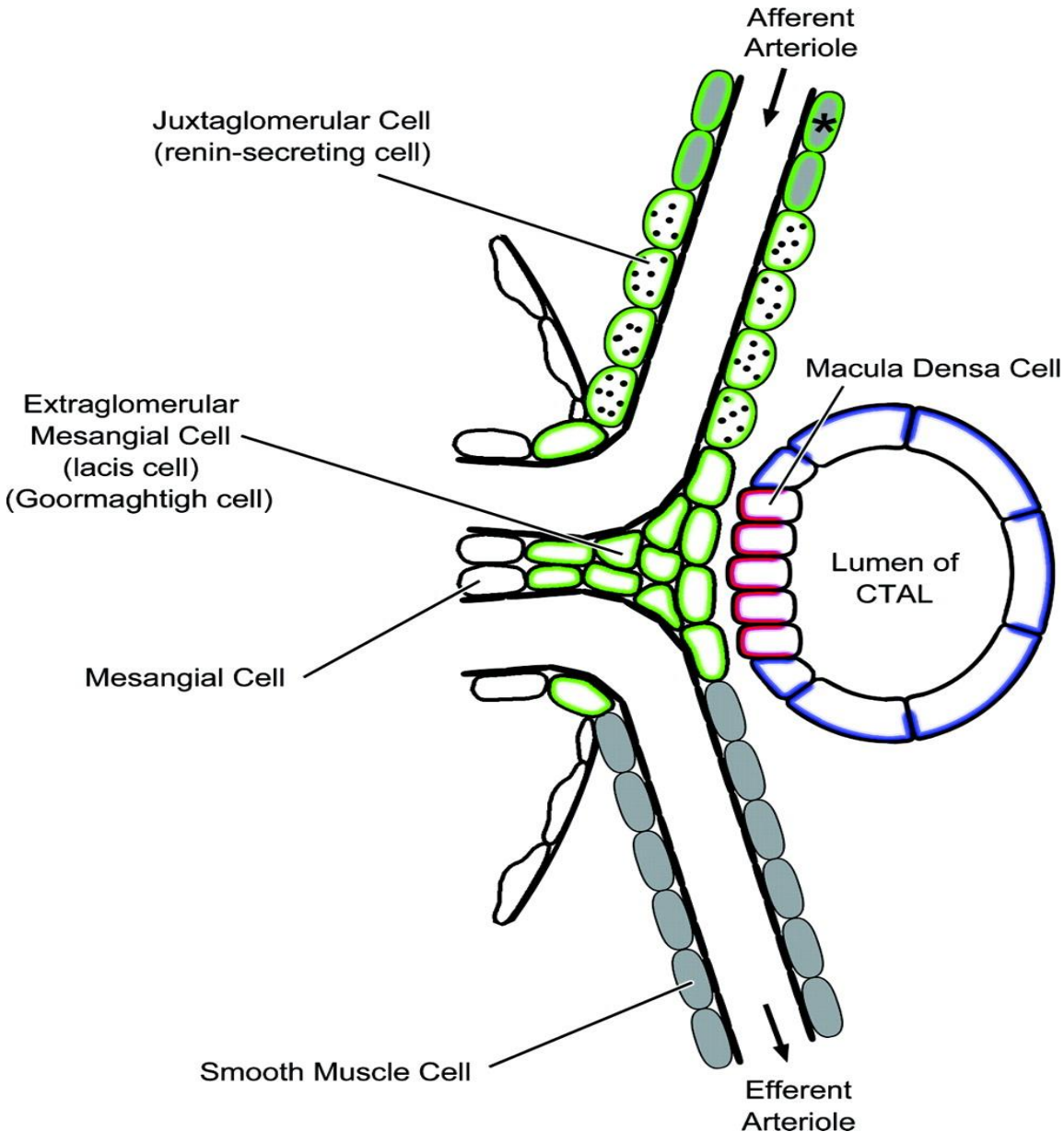
1. Postural changes,
2. Emotional stress
3. Physiologic stress
4. Blood volume changes

The baroreflex activity declines during sustained increase in pressure. Patients with dysautonomia and impaired baroreflex mechanism may develop extreme labile blood pressures associated with tachycardia.

Renin-Angiotensin-Aldosterone system

The renin-angiotensin-aldosterone system regulates of arterial pressure mainly by

1. Angiotensin II
2. Aldosterone.



Renin is an

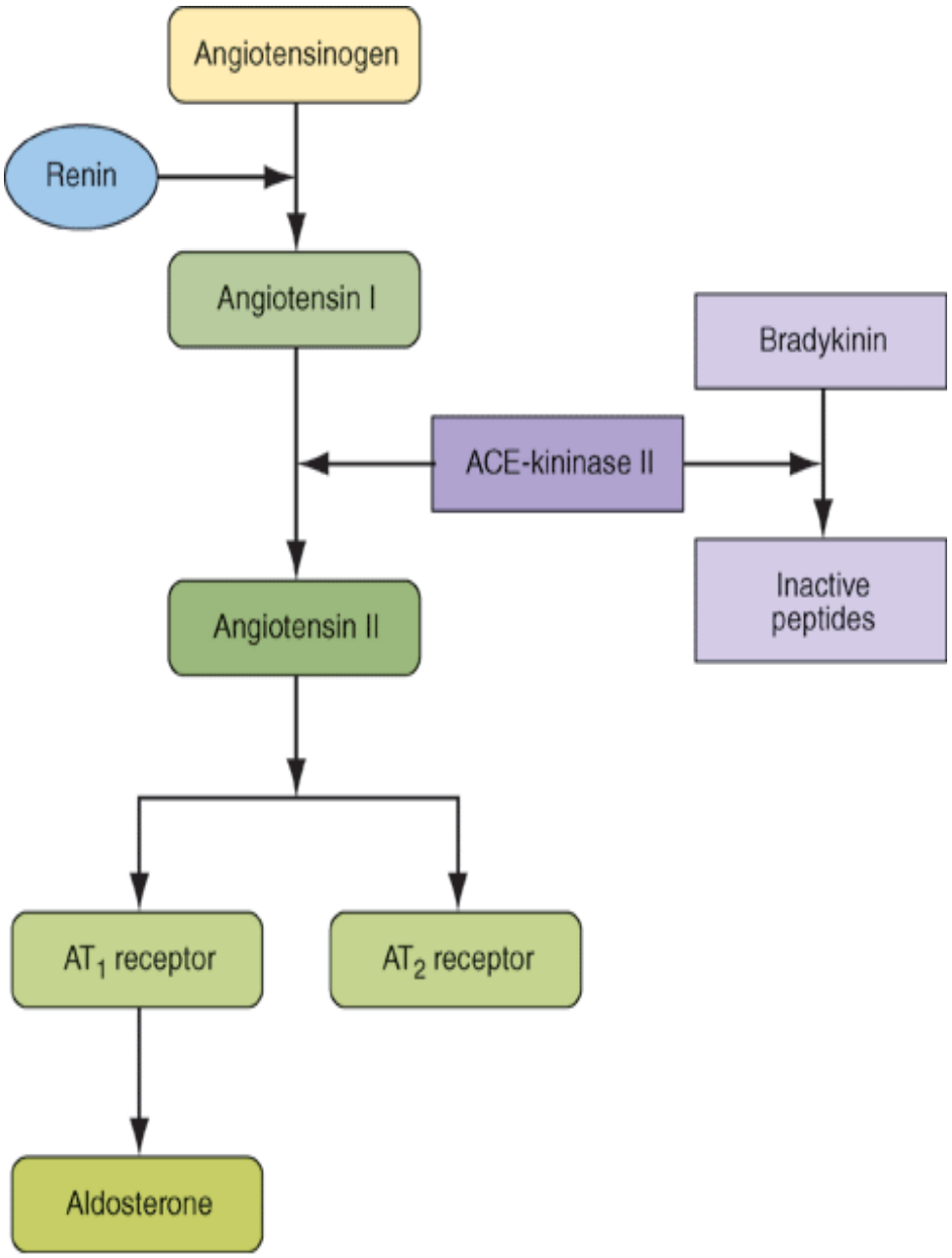
1. Aspartyl protease synthesized from inactive precursor prorenin.
2. It is synthesized from the renal afferent renal arteriole.
3. Prorenin is secreted directly into the circulation.

The activators of renin secretion:

1. Decreased Na transport in the loop of Henle
2. Decreased pressure in renal afferent arteriole.
3. Sympathetic nervous system.

Renin inhibitor

1. Increased Na transport in the loop of Henle
2. Increased pressure in the afferent arteriole
3. Alpha 1 receptor blockade.
4. Angiotensin II.



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Angiotensin II is a

1. Potent pressor substance
2. Tropic factor for the secretion of aldosterone frome adrenal zona glomerulosa.
3. Potent mitogen stimulating vascular smooth muscle cell and myocytes
4. Angiotensin II contributes to atherosclerosis

Aldosterone is regulated by

1. Angiotensin II
2. Potassium level
3. Acute elevations of adrenocorticotropic hormone

Aldosterone is a mineralocorticoid that regulates

1. Amiloride sensitive epithelial sodium channels.
2. Hydrogen potassium pump. Increased aldosterone secretion results in alkalosis and hypokalemia.

Mineralocorticoid receptors expressed in the sweat glands, colon and salivary glands. Aldosterone also has effects on other targets.

Aldosterone receptor activation induces structural and functional changes in

1. Heart – Myocardial fibrosis
2. Kidney – Nephrosclerosis
3. Blood vessels – vascular inflammation and remodelling

In heart failure patients, low-dose spironolactone decreases the risk of progressive heart failure and sudden death by 30%. Aldosterone may reduce glomerular hyper filtration and albuminuria.

Vascular Mechanism

Radius of blood vessel and compliance in arteries are important determining factors of arterial pressure.

Resistance to flow varies inversely with the radius, and therefore, small decreases in lumen size increase the resistance.

In hypertensive persons, structural, functional and mechanical changes reduce the lumen of small arteries and arterioles.

Hypertrophic changes are due to

1. Low-grade inflammation
2. Cell size increase

3. Increased deposition of intercellular matrix

4. Vascular fibrosis

Diameter of the lumen is related to vessel wall elasticity. Vessels with increased elasticity can accommodate an increased volume with little change in pressure and vessel with less degree of elasticity, a small increase in volume produces a large increase in pressure.

Arteriosclerotic patients usually have high SBP and wide pulse pressure as a result of decrease in vascular compliance. Recent trails suggest that arterial vessel wall stiffness is a predictive factor for cardiac events. The devices are ultrasound and magnetic resonance imaging, used to measure arterial stiffness.

Ion transport in vascular smooth muscle cells may contribute to hypertension. The ion channels include

1. Na^+ dependent HCO_3^--Cl^- exchange

2. Cation independent $HCO_3^-Cl^-$ exchange

3. Na^+ H^+ exchange.

Vascular endothelial function contributes to tone of vessel wall. The vascular endothelium releases nitric oxide. It is a vasodilator.

Endothelium-dependent vasodilation is impaired in hypertensive patients. It is assessed with ultrasonography after occluding the vessel wall for 5 minutes then measuring the perfusion in the hyperemic phase in the forearm. It can also be tested directly by infusing the acetyl choline.

Endothelin, it is a vasoconstrictor substance released by endothelium. Increased production can lead to increase in blood pressure. Endothelin antagonists can decrease blood pressure.

Pathologic Consequences of Hypertension

Heart

Cardiac disease is one of the common cause of death in patients with elevated blood pressure

Hypertension can lead to

1. Left ventricular hypertrophy
2. Congestive cardiac failure
3. Microvascular disease
4. Arrhythmias

5. Atherosclerotic coronary artery disease

Left ventricular hypertrophy is usually diagnosed by electrocardiography. The echocardiography is sensitive for assessment of thickness of left ventricular wall.

Individuals with LVH are at increased risk for coronary artery disease, stroke, congestive cardiac failure and sudden death. The reversal of hypertension can reverse the hypertrophy of left ventricle and can decrease the risk of heart disease.

Congestive heart failure may be because of systolic dysfunction or diastolic dysfunction, or a combination of both. The diastolic dysfunction ranges from asymptomatic disease to severe heart failure.

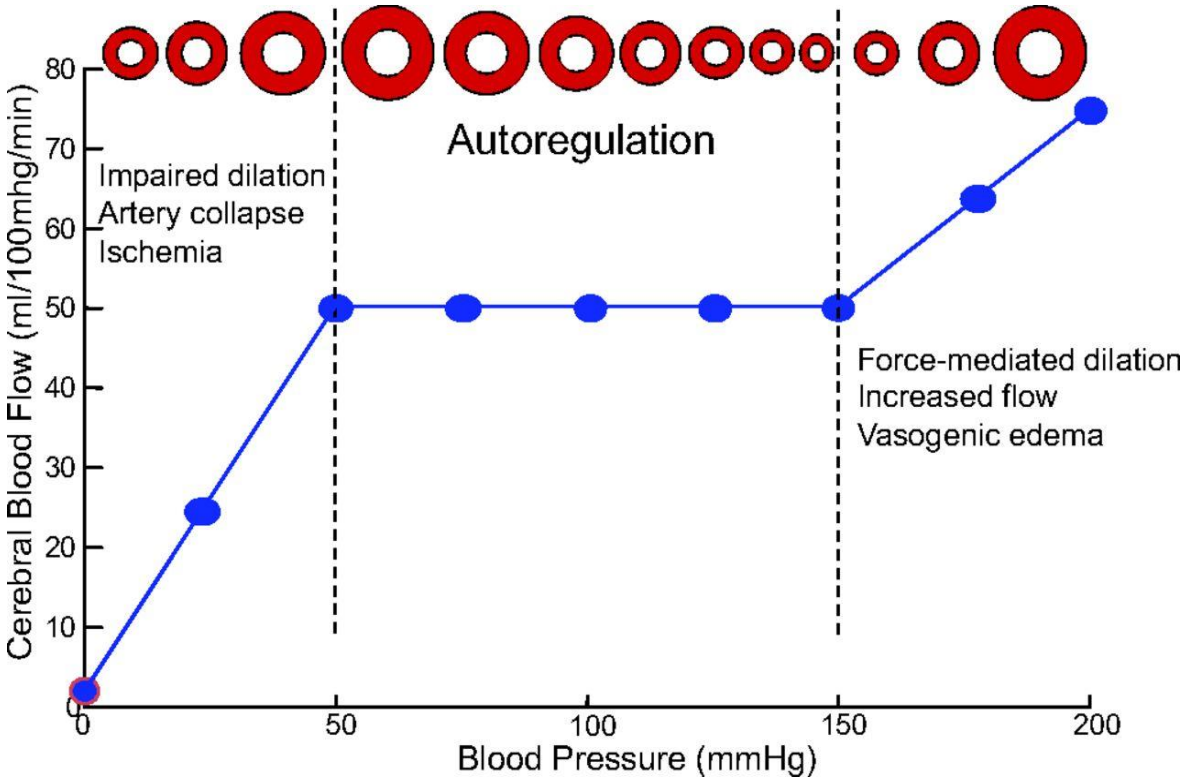
Patients with diastolic dysfunction usually have a normal ejection fraction. In 1/3 of patients with CHF, the function of systole is normal and it is mainly due to diastolic dysfunction. Diastolic dysfunction occurs early in hypertensive disease and is increased by hypertrophy of left ventricle and ischemia. Cardiac catheterization gives the most accurate measurement of diastolic dysfunction. It can be evaluated by also echocardiography and radionuclide angiography.

Brain

Increased blood pressure is a risk factor for stroke. In stroke 85% of it, is due to infarction, and the 15 % are due to haemorrhage. The incidence of stroke increases with elevated blood pressure and the treatment of hypertension decrease the incidence of stroke.

Hypertension can also due to impaired cognition in an elderly population. Hypertension related dementia and cognitive impairment may be a

1. Due to single infarct of larger vessel
2. Multiple lacunar infarcts of small vessel disease



Kidney

The renal diseases are the most common cause for secondary hypertension. Hypertension is a risk factor for renal disease and can lead to ESRD. The more the increase in blood pressure, more damage to kidneys over long time and it is mostly related to systolic blood pressure.

Proteinuria is a marker for severity of chronic kidney disease and it also predicts the progression of renal disease. Patients with heavy protein excretion of more than three grams per day have a more rapid rate of progression.

In hypertension, the kidney losses auto regulation of blood flow at the afferent arteriole which results in elevated pressure in unprotected glomerulus leading to glomerular hyper filtration, hypertrophy and glomerular sclerosis.

This leads to vicious cycle of kidney damage and nephron loss leading to worsening of hypertension, glomerular hyper filtration, and more renal damage. Macro albuminuria is defined as urine albumin to creatinine ratio more than >300 mg/g and micro albuminuria is defined as urine albumin to creatinine ratio 30–300 mg/g. Micro and macro albuminuria is a risk factor cardiovascular disease.

Peripheral Arteries

The blood vessels are a target organ for atherosclerotic disease and also for elevated blood pressure. Majority are asymptomatic and patients can present with intermittent claudication which can present like aching pain in the calve muscles and buttocks exaggerated by walking and relieved by taking rest.

The ankle-brachial index is defined as ankle to brachial systolic blood pressure ratio measured noninvasively.

An ankle-brachial index less than 0.90 is diagnostic of peripheral arterial disease and it can be associated with more than 50% stenosis of at least one lower limb vessel.

Hypertensive emergencies

Definition

According to JNC 7 report on prevention, evaluation and treatment of blood pressure.

Hypertension is defined as systolic blood pressure ≥ 140 or a diastolic blood pressure ≥ 90 in patients with known hypertension or measured in two or more settings.

Blood pressure classification	SBP mm Hg	DBP mm Hg
Normal	<120	And <80
Prehypertension	120 – 139	80 – 89
Stage 1 hypertension	140 – 159	90 – 99
Stage 2 hypertension	≥160	≥100

Patients with systolic blood pressure greater than 179 or diastolic blood pressure greater than 119 are usually defined as having hypertensive crisis

Hypertensive crisis

Severe alteration in blood pressure that have potential to cause target organ damage. The target organ includes brain, eyes, heart, blood vessel and kidney. Hypertensive crisis include hypertensive urgency and emergency.

Hypertensive urgency

Hypertensive urgency characterised by severe elevation in blood pressure without progressive target organ damage.

Hypertensive emergency

Hypertensive emergency characterised by severe elevation in blood pressure (>180/120) with progressive target organ dysfunction.

This requires immediate reduction in blood pressure to prevent or limit target organ damage.

Differentiating these two entities is important in treatment aspect because in HE blood pressure should be lowered immediately but not to normal level and in hypertensive urgency blood pressure should be reduced over 24 – 48 hours

Accelerated hypertension

It is defined by Keith and Wagner, characterised by severe elevation in BP in the presence of haemorrhage in retina and also the presence of exudates in the absence of papilledema

Malignant hypertension

It is defined by Volhard and Fahr, chararcterised by severe elevation in BP associated with retinopathy, papilledema, renal failure and renal arteriole fibrinoid necrosis.

Currently all these term are replaced by the term hypertensive urgency and emergency

Before the introduction of anti hypertensive drug nearly 7% of hypertensives developed hypertensive crisis and the mortality were high. Now after the introduction of parentral drugs the mortality has come down but still it is common.

Before diagnosing hypertensive emergency the other close mimics should be ruled out. These are called hypertensive pseudo crisis which includes transient elevation blood pressure due to

1. Pain
2. Hypoxia
3. Emotional stress
4. Vertigo
5. Spinal cord injury
6. Acute urinary retention

In these conditions blood pressure will come down once the cause is treated.

Epidemiology of hypertensive emergencies

In India, the elderly population has increased and the number of people more than 65 years of age is expected to increase in next 2 – 3 decades. This will lead to increase in prevalence of hypertension in India.

The prevalence of hypertension ranges from 20 – 40 % in urban India and 12 – 17 % in rural India.

With the development of anti hypertensive medication the estimated prevalence is that 1 -2 % of patients with hypertension will have HE at some point during their lifetime

Etiology

Hypertensive emergency can develop from essential hypertension or due to secondary hypertension. It most commonly arises from essential hypertension and kidney disease is the common cause in secondary cause for hypertension. The secondary cause includes

Causes for hypertensive emergency	
1	Essential hypertension
2	Renal parenchymal disease 1. Diabetes mellitus 2. Glomerulonephritis 3. Tubulointerstitial diseases 4. HUS 5. TTP 6. SLE 7. Systemic sclerosis 8. Renal cell carcinoma 9. Renal aplasia
3	Renovascular diseases 1. Renal artery stenosis

	2. Polyarteritis nodosa 3. Takayasu arteritis
4	Endocrine causes 1. Cushing syndrome 2. Pheochromocytoma 3. Renin secreting tumour 4. Primary hyperaldosteronism
5	Autonomic hyperreactivity 1. GBS 2. Acute porphyria
6	Pregnancy related 1. Preeclampsia 2. Eclampsia
7	Central nervous system 1. Head injury 2. Cerebral infarction 3. Cerebral haemorrhage 4. Brain tumour 5. Spinal cord injury
8	Drugs 1. Sympathomimetics

	2. Cocaine 3. Amphetamine 4. MAO inhibitor 5. Erythropoietin 6. Cyclosporine 7. Anti hypertensive drug withdrawal
9	Burns
10	Coarctation of aorta
11	Post operative/anaesthetic complications

Pathophysiology

Sudden increase in vascular resistance is related to vasoconstrictor, renin angiotensin system and inflammatory cytokines.

The increase in BP leads to increase in mechanical stress and also endothelial injury leading to

1. Increase in permeability
2. Coagulation pathway activation
3. Platelet activation
4. Fibrin deposition

This leads to ischemia and more release of vasoactive substance resulting in vicious cycle, leading to end organ damage.

Thrombotic microangiopathy is characterised by endothelial dysfunction, activation of platelet and thrombin generation.

Volume depletion resulting from natriuresis stimulates the release of vasoconstrictors from kidney, these substances further adds to the damage.

Clinical manifestation

The various manifestations of hypertensive emergency includes

1. Hypertensive encephalopathy
2. Cerebral infarction
3. Subarachnoid haemorrhage
4. Intracranial haemorrhage
5. Acute myocardial infarction
6. Acute LVF
7. Acute pulmonary edema
8. Dissection of aorta
9. Ecclampsia
10. Hypertensive retinopathy
11. Renal insufficiency
12. Microangiopathic haemolytic anemia

Neurologic syndromes

The sudden severe elevated blood pressure leads to cerebral edema and micro haemorrhages in brain. The symptoms include

1. Headache
2. Nausea/vomiting
3. Visual symptoms

4. Delirium
5. Stupor
6. Seizures
7. Focal neurological deficits

Cardiac syndromes

There is an increase in myocardial wall tension and oxygen demand, it can lead to myocardial ischemia and infarction. The development of pulmonary edema is due to mainly diastolic dysfunction. The symptoms include

1. Chest pain
2. Breathlessness
3. Cough
4. Abdominal pain
5. Signs of poor perfusion

Aortic dissection

Aortic dissection is rare but dreaded complication of hypertensive crisis. It is often misdiagnosed. It usually presents with rapid onset chest pain radiating to back, tearing in nature. Majority are previously diagnosed as hypertensives

Renal syndromes

The symptoms include

1. Oliguria
2. Anuria
3. Nausea and vomiting
4. Peripheral edema
5. Altered mental status

Hypertension in pregnancy

It occurs in 5 -7 % of pregnancy. Blood pressure of > 140/90 after 20 weeks of pregnancy, characterised by proteinuria and multi organ involvement

Post operative hypertension

It is defined as severe elevation in blood pressure occurring within 2 hours of surgery, due to sympathetic activation and adrenergic surge. Causes for sympathetic activation includes

1. Pain
2. Anxiety
3. Hypoxia
4. Hypercarbia
5. Hypothermia
6. Volume overload
7. Urinary retention

8. Drug withdrawal

Hypertensive encephalopathy

It is diagnosis of exclusion, after excluding cerebral haemorrhage and ischemic stroke. There will not be any neurological deficit in encephalopathy

Evaluation of hypertensive emergencies

The important aspect in managing hypertensive emergency is differentiating it from hypertensive urgency which does not require aggressive blood pressure reduction. It requires complete history, physical examination and investigations

History

1. Symptoms related to target organ damage
2. Hypertension history
    a. Last known BP
    b. Dietary adherence
3. Cardiac history
    a. Previous myocardial infarction/angina pain
    b. Previous arrythmias
4. Neurologic history
    a. Previous stroke
    b. Previous neurological dysfunction
5. Renal history
    a. History previous renal disease
    b. History of proteinuria
6. Endocrine history
    a. Diabetes
    b. Thyroid

    c. Cushing syndrome

7. Personal history
8. Family history
9. Drug history
    a. Duration of drug intake
    b. Compliance with drug
    c. Adequate control in past
    d. Dosing of drugs
    e. Previous crisis
    f. Any recent change in drugs

Examination

Measurement of blood pressure

Cardiovascular system

Pulse in all extremities

Jugular venous pulse

Third heart sound

Heart murmur

Respiratory system

Bilateral crepitation

Abdomen

Auscultation for renal bruit

Central nervous system

Level of consciousness

Presence of focal neurological deficit

Fundoscopic examination

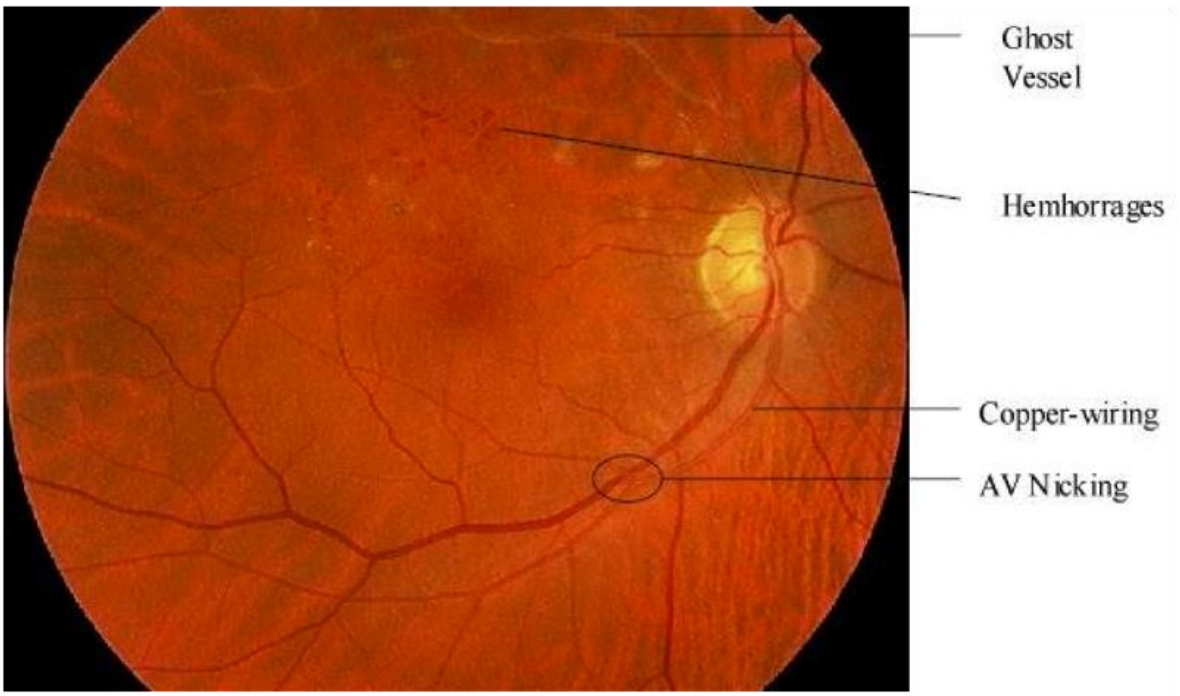
Keith Wagner Barker grading

Grade 1 – Mild generalised arteriolar narrowing

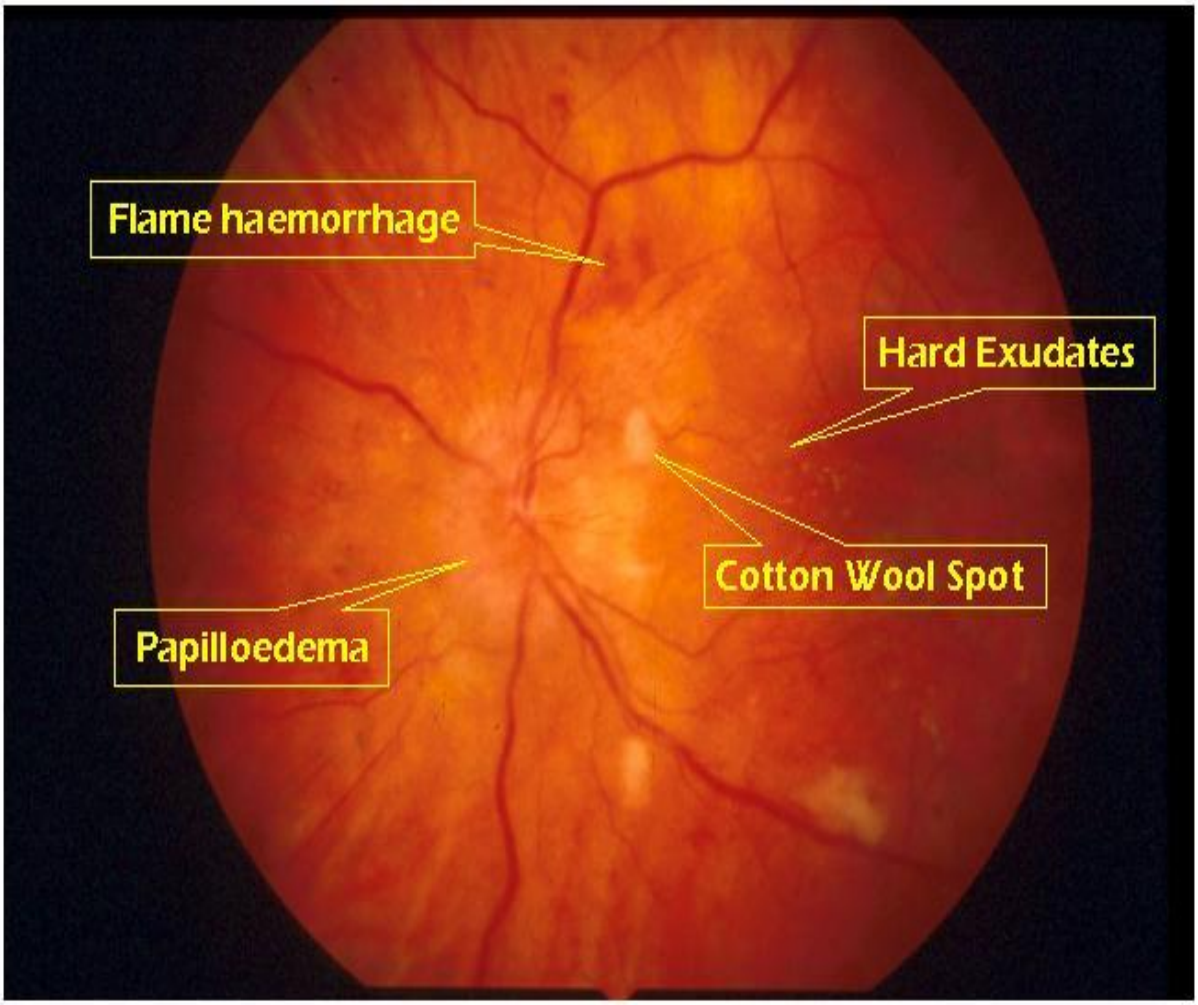
Grade 2 – Focal narrowing and arteriovenous nipping

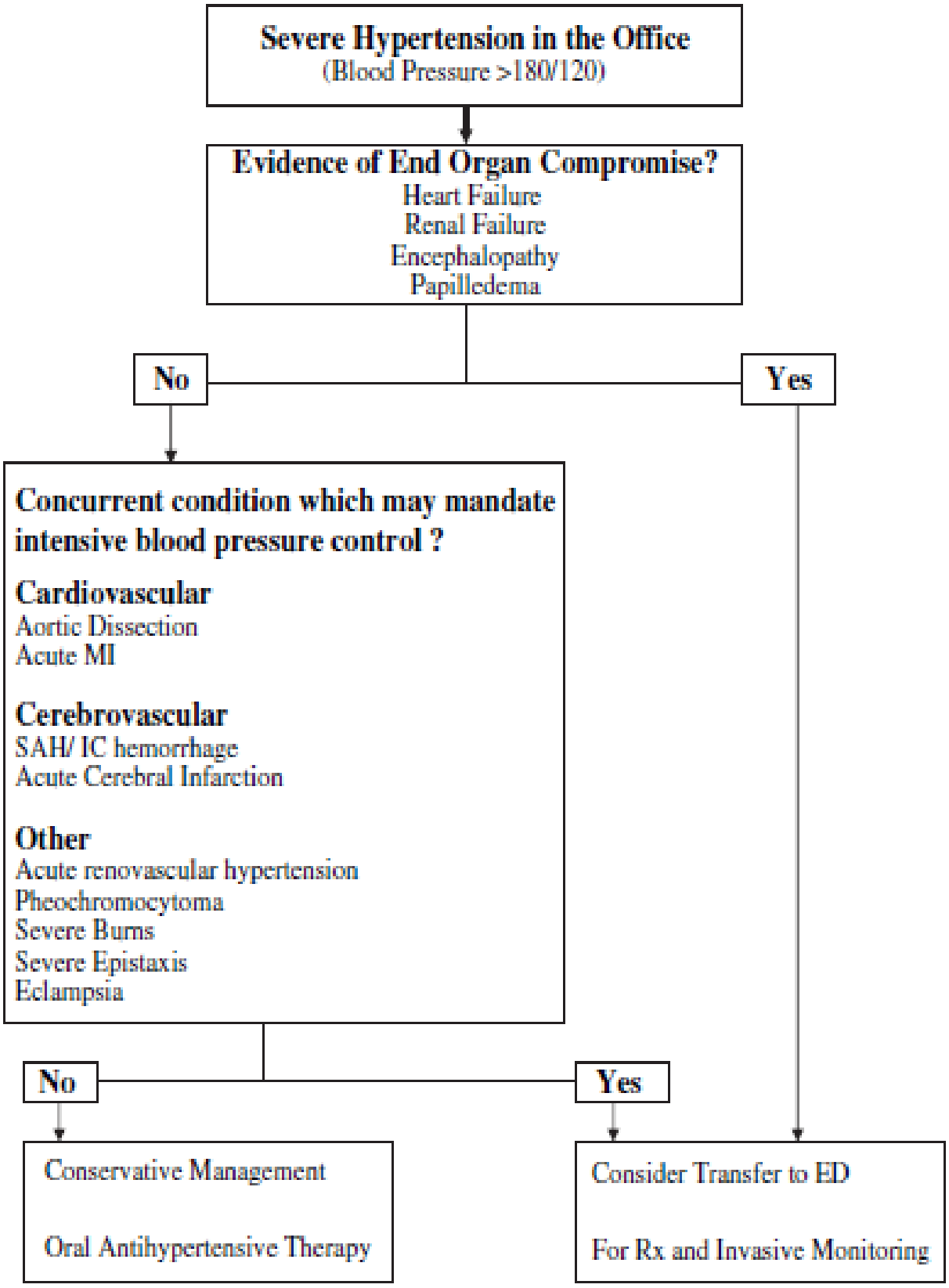
Grade 3 – Grade 2 changes plus retinal haemorrhages, exudates and cotton wool spots. Associated with cardiac, CNS and kidney compromise.

Grade 4 – Grade 3 plus papilloedema. Organ compromise is more severe.



Hypertensive Retinopathy - Grade 4




Severe Hypertension in the Office
(Blood Pressure >180/120)
Evidence of End Organ Compromise?
Heart Failure
Renal Failure
Encephalopathy
Papilledema
No
Yes
Concurrent condition which may mandate intensive blood pressure control ?
Cardiovascular
Aortic Dissection
Acute MI
Cerebrovascular
SAH/ IC hemorrhage
Acute Cerebral Infarction
Other
Acute renovascular hypertension
Pheochromocytoma
Severe Burns
Severe Epistaxis
Eclampsia
No
Yes
Conservative Management
Oral Antihypertensive Therapy
Consider Transfer to ED
For Rx and Invasive Monitoring

Investigations

1. Renal function test
    a. Urea
    b. Creatinine
2. Serum electrolytes
    a. Na
    b. K
3. Complete hemogram
    a. Hemolytic anaemia
    b. Low platelet count
4. Reticulocyte count
5. Lactate dehydrogenase
6. Electrocardiogram
    a. Myocardial infarction
    b. Unstable angina
    c. Hypertensive changes
7. Urine analysis
    a. Proteinuria
    b. Haematuria
8. X ray chest
    a. Cardiomegaly

    b. Pulmonary edema

9. CT brain
    a. Infarct
    b. Haemorrhage
10. Echo cardiography
    a. Systolic dysfunction
    b. Diastolic dysfunction

Special situation

1. Aortic dissection
    a. Trans esophageal echocardiography
    b. Contrast CT chest
    c. MRI
2. Subarachnoid haemorrhage
    a. Lumbar puncture – RBC'S
3. Sympathetic crisis
    a. Urine catecholamine
    b. Urine metanephrine
4. Recreational drugs
    a. Urine toxicology

Based on the history, examination and relevant investigations, the patient can be classified as having hypertensive urgency or emergency and the treatment should be initiated accordingly.

Approach

As mentioned in the previous discussion, the patient should be classified as having hypertensive urgency or emergency.

In hypertensive urgency, using oral drugs the BP is lowered gradually over 24 – 48 hours

Rapid reduction of BP in these patients may lead to morbidity due to alteration in auto regulation of blood pressure due to chronicity of blood pressure.

It can lead to reduction in perfusion causing ischemia and infarction. It must be reduced in a controlled fashion to avoid organ hypoperfusion

Patient with hypertensive emergency is best managed with short acting anti hypertensive agent by continuous infusion.

These patients are closely monitored in an intensive care unit. It is advised not to treat these patients outside intensive care setup, because there may be rapid fall in blood pressure which can lead to morbidity and mortality. Invasive arterial blood pressure monitoring can be used in patients with severe clinical manifestations

The goal is to reduce MAP by 20 – 25 % within minutes to hours and then to 160/110 in next 5 hours. When the BP is under control, oral therapy should be initiated and the parenteral drug should be slowed down

Patient with hypertensive emergency may be depleted in volume and there may be rapid precipitous fall in BP when hypertensive drug is initiated due to pressure natriuresis. It is better to avoid nitroglycerin except in cardiac cases.

In stroke patients hypertension may be protective and further reducing the BP may be harmful. In these patients there is no evidence that increased blood pressure is harmful. Infact reducing the blood pressure has resulted in deleterious effects in some patients.

In patients with cerebral infarction, the guideline is to initiate antihypertensive therapy when SBP > 220 or DBP > 130.

In patients with haemorrhagic stroke, the guideline is to initiate antihypertensive therapy when SBP > 180 or DBP > 130.

In pregnancy SBP > 180 or DBP > 110, parenteral drugs may be used but it is desirable not to reduce DBP < 90 because of placental perfusion.

Management

Drugs considered in hypertensive emergencies	
Cerebral infarction	1. Labetolol 2. Nicardipine 3. Fenoldopam
Intracerebral haemorrhage	1. Labetolol 2. Nicardipine
Hypertensive encephalopathy	1. ACE inhibitor 2. Labetolol 3. Nicardipine
Acute coronary syndrome	1. Nitroglycerin 2. Labetolol 3. Esmolol 4. ACE inhibitor
Acute pulmonary edema	1. Nitroglycerin 2. Nitroprusside 3. Loop diuretic
Aortic dissection	1. Labetolol 2. Esmolol 3. Fenoldopam

	4. Nitroprusside
Eclampsia	1. Magnesium sulphate 2. Labetolol
Acute renal failure	1. Labetolol 2. Nicardipine 3. Fenoldopam

The blood pressure goals considered in specific situations

Blood pressure goals		
1	Hypertensive encephalopathy	MAP lowered by 20 - 25 %
2	Ischemic stroke	Controversial Reduce BP SBP > 220, DBP > 130
3	Haemorrhagic stroke	Controversial Reduce BP SBP > 180, DBP >130
4	Hypertensive retinopathy	MAP lowered by 20 - 25 %
5	Left ventricular failure	MAP reduction to 60 – 100 mm Hg
6	Myocardial ischemia	MAP reduction to 60 – 100 mm Hg
7	Aortic dissection	SBP 100 – 120 mm Hg
8	Renal insufficiency	MAP lowered by 20 - 25 %
9	Post-operative hypertension	MAP lowered by 20 - 25 %
10	Pregnancy induced hypertension	SBP 130 – 140 mm Hg, DBP 80 – 100 mm Hg
11	Hyper adrenegic states	MAP lowered by 20 - 25 %

Pharmacologic agents

Oral drugs

Labetolol

Combined alpha and beta adrenergic blocking agent

Orally 200 – 400 mg

Onset of action 1- 2 hrs

Avoid in asthmatics, bradycardia, heart block and congestive heart failure

Prasozin

Alpha adrenergic blocking agent

Can cause first dose syncope, palpitations, tachycardia and orthostatic hypotension

Captopril

ACE inhibitor

Onset of action 10 – 20 mins

Can cause renal failure in bilateral renal artery stenosis and reflex tachycardia

Clonidine

Centrally acting alpha agonist

Onset of action 30 – 60 mins

Can cause light headedness, drowsiness and dry mouth

Avoid in bilateral renal artery stenosis

Parenteral drugs

Labetalol

Mechanism of action: combined alpha and beta blocking agent

Onset: 2- 5 minutes

Peak: 15 minutes

Duration: 2-4 hrs

Dosage: bolus 10 -20 mg, continuous 3- 10 mg/min

Avoid: Asthmatics, bradycardia, heart block and congestive heart failure

Caution: Liver failure, COPD and elderly

Esmolol

Mechanism of action: Cardio selective beta blocker

Onset: 60 seconds

Duration: 10 – 20 minutes

Dosage: Bolus: 500 microgram/kg, Continuous: 25–300 microgram/kg/min

Avoid: Asthmatics, bradycardia, heart block and congestive heart failure

Caution: Extravasation can lead to skin necrosis and sloughing

Nicardipine

Mechanism of action: Dihydropyridine calcium channel blockers

Onset: 5 – 10 minutes

Duration: 1- 4 hours

Dosage: Start at 5mg/hr, then 2.5 mg/hr infusion

Avoid: Patients receiving intravenous beta blockers

Caution: decompensated heart failure

Nitroglycerin

Mechanism of action: Direct venodilator

Onset: 2 minutes

Duration: 1 hour

Dosage: Start at 5 mcg/min up to maximum 200 mcg/min

Avoid: Concomitant use with phosphodiesterase 5 inhibitor, impaired cerebral and renal perfusion

Caution: Hypotension and reflex tachycardia

Nitroprusside

Mechanism of action: Arterial and venous dilator due to nitric oxide

Onset: seconds

Duration: 1- 2 minutes

Dosage: 0.5 mcg/kg/min up to 2 mcg/kg/min

Avoid: Renal failure, cardiac failure and increased intracranial pressure

Caution: Coronary steal syndrome, Intra arterial BP monitoring is required

Phentolamine

Mechanism of action: Alpha 1 and 2 blocking action

Dosage: 5 mg over 5 minutes, then 0.5 mg/min

Caution: Cerebrovascular accident, Myocardial infarction

Fenoldopam

Mechanism of action: Dopamine 1 receptor agonist

Onset: 5 minutes

Peak: 15 minutes

Duration: 60 minutes

Dosage: 0.1 mcg/kg/min, maximum 1.6 mcg/kg/min

Caution: Reflex tachycardia, giddiness and vomiting

Enalaprilat

Mechanism of action: Angiotensin converting enzyme inhibitor

Dosage: 1.25 mg over 5 minutes, maximum 5 mg every 6 hours

Avoid: Pregnancy

Caution: First dose hypotension, headache

Diazoxide

Mechanism of action: Potassium channel opener

Onset: 2 -5 minutes

Duration: 3 – 12 hrs

Dosage: 50 mg iv bolus, then 15 mg/min iv infusion

Caution: Hypotension, tachycardia, nausea, vomiting and angina

Hydralazine

Mechanism of action: Directly acting vasodilator

Onset: 10 minutes

Duration: 60 minutes

Dosage: 5 mg intra venous bolus, followed by 10 – 40 mg every 4 – 6 hrs

Caution: Tachycardia, headache and vomiting

Objectives

1. **To assess the prevalence of hypertensive emergency among the hospital admission over the period of 2 months**

2. **To assess the risk factors, clinical presentation and profile of patients in hypertensive emergencies**

4. Materials and methods

Study design

Cross sectional descriptive hospital based study

Study participants

Patients admitted in admitting unit and medicine ward of Stanley medical college

Study population

Adults more than 18 years of age

Study period

June 2014 to August 2014

Inclusion criteria

1. Age > 18 years.
2. severe elevations in BP (>180/120 mmHg) associated with evidence of progressive target organ damage at the time of admission(JNC 7 criteria)
3. Willing to participate and able to give valid consent.

Exclusion criteria

1. Age < 18 years
2. Severe elevations in BP without progressive target organ dysfunction
3. Not willing to participate.

Sample size

All the patients fulfilling the inclusion criteria admitted in hospital will be studied for 2 - 3 months.

Methodology

The study will be carried out in all patients more than 18 years of age and hypertensive emergencies admitted to medical ward in Stanley medical college from June 2014 to August 2014. On admission detailed history, examination and relevant investigations will be done.

Stasistics

All the data will be entered in a master excel sheet and will be analysed using SPSS software. Categorical variables will be expressed as absolute (n) and relative (%) frequencies and continuous variables will be expressed as means and standard deviation.

Results and discussion

Prevalence

In our study there were 82 participants over the period of two months. Based on the number of admissions for two months the prevalence of hypertensive emergencies is in our setup is 0.687

Table 1 Profile of study participants presented with Hypertensive emergencies. (N=82)

S.No.	Profile	N(%)
1.	**Age (in years)**	
	30-45	8(9.8)
	45-60	40(48.8)
	>60	34(41.5)
2.	**Gender**	
	Male	43(52)
	Female	39(48)

This study contains 82 subjects, observed over the period of two months. In our study group 43 were male and 39 were female and the corresponding percentage was 52 % and 48 %.

The minimum age was 31 and maximum was 82. The mean age is 59.

Our study group contained 41 % in elderly group, 49 % between age of 45 to 60 and nearly 10 % below the age of 45

Figure 1. Male female ratio of study participants with hypertensive emergencies.

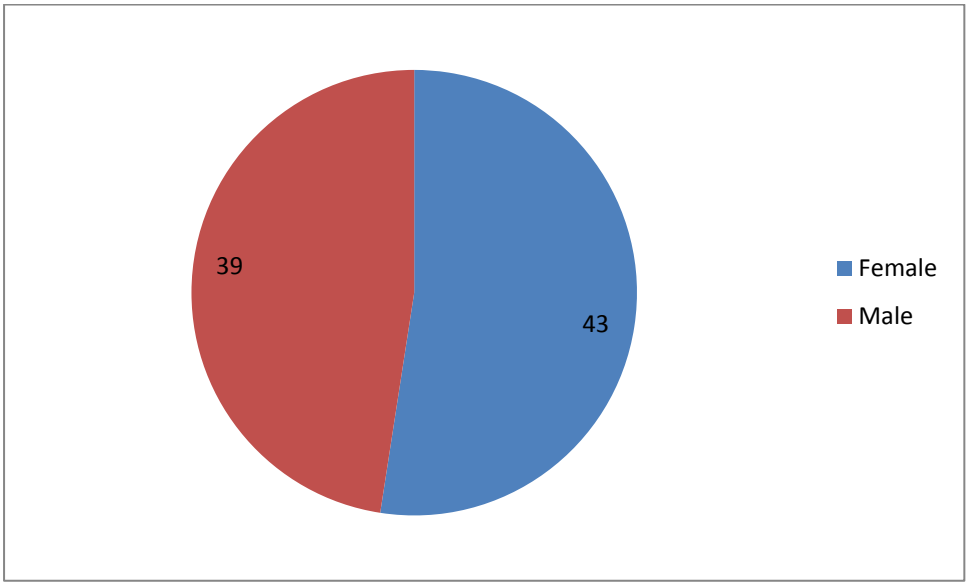


Figure 2. Age group of study participants with hypertensive emergencies.

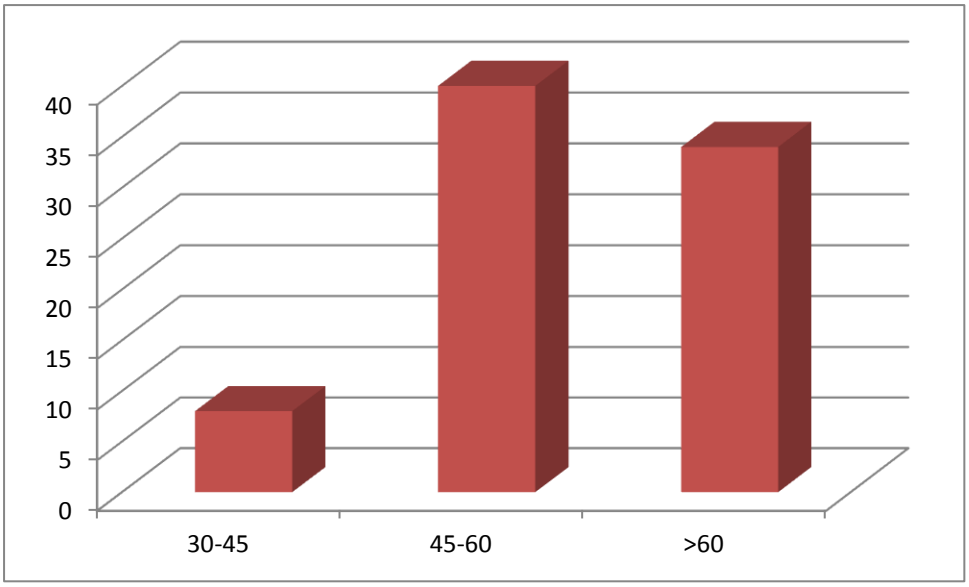


Table 2. Prevalence of presenting complaints of Hypertensive emergencies. (N= 82)

S. No.	Presenting Complaints*	N (%)
1.	Headache	33(40.2)
2.	Giddiness	33(40.2)
3.	Neurological deficit	22(26.0)
4.	Breathlessness	18(21.9)
5.	Chest pain	18(21.9)
6.	Altered mental status	12(14.6)
7.	Visual changes	10(12.0)
8.	Nausea/Vomiting	9(10.9)
9.	Seizure	8(9.7)
10.	Epistaxis	1(1.2)

*** Multiple response**

The most common presenting symptoms were predominantly neurological which includes headache, giddiness and neurological deficit.

The second most common presenting symptoms were from cardiovascular system, includes chest pain and breathlessness

Figure 3. Prevalence of presenting complaints of Hypertensive emergencies.

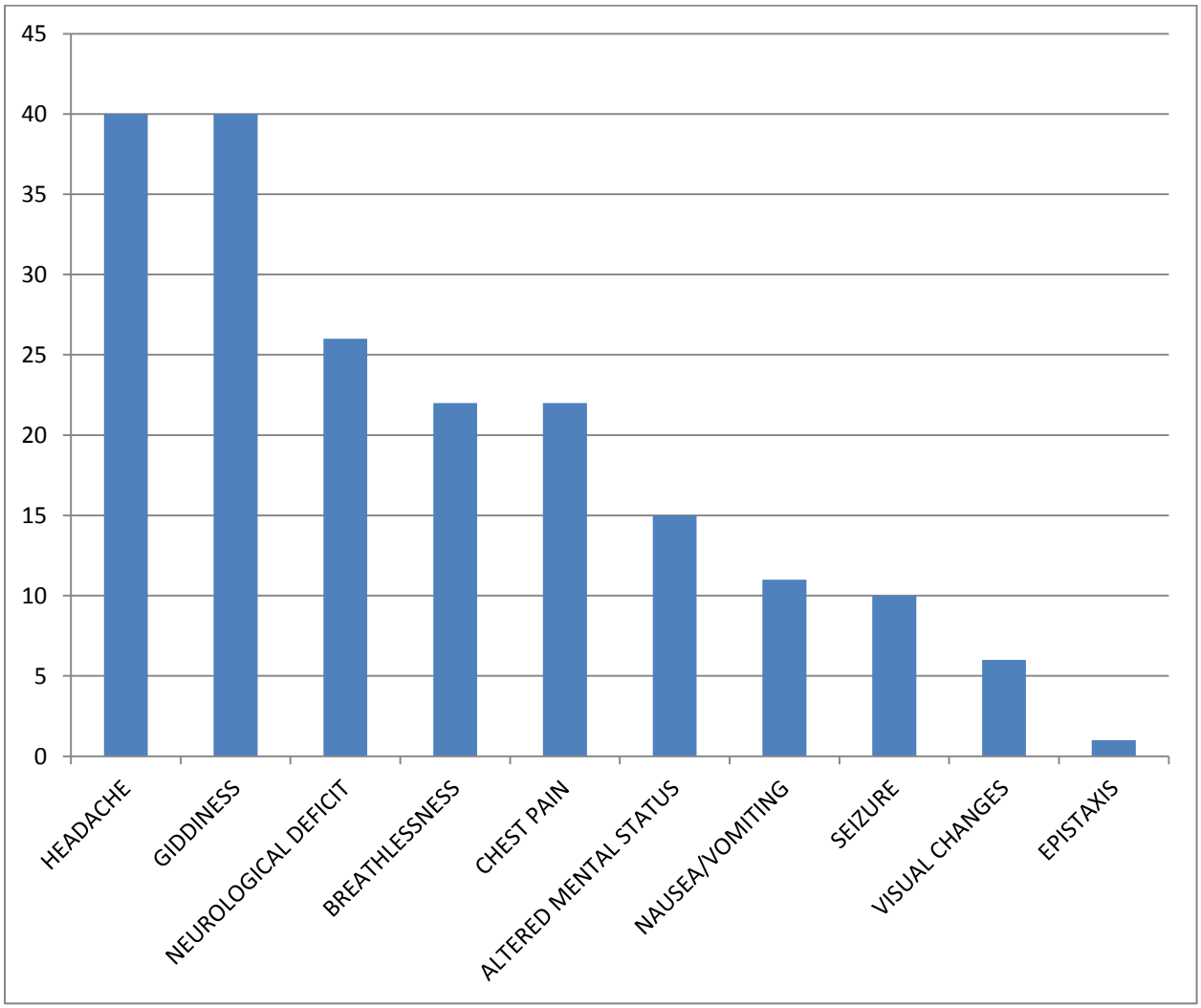


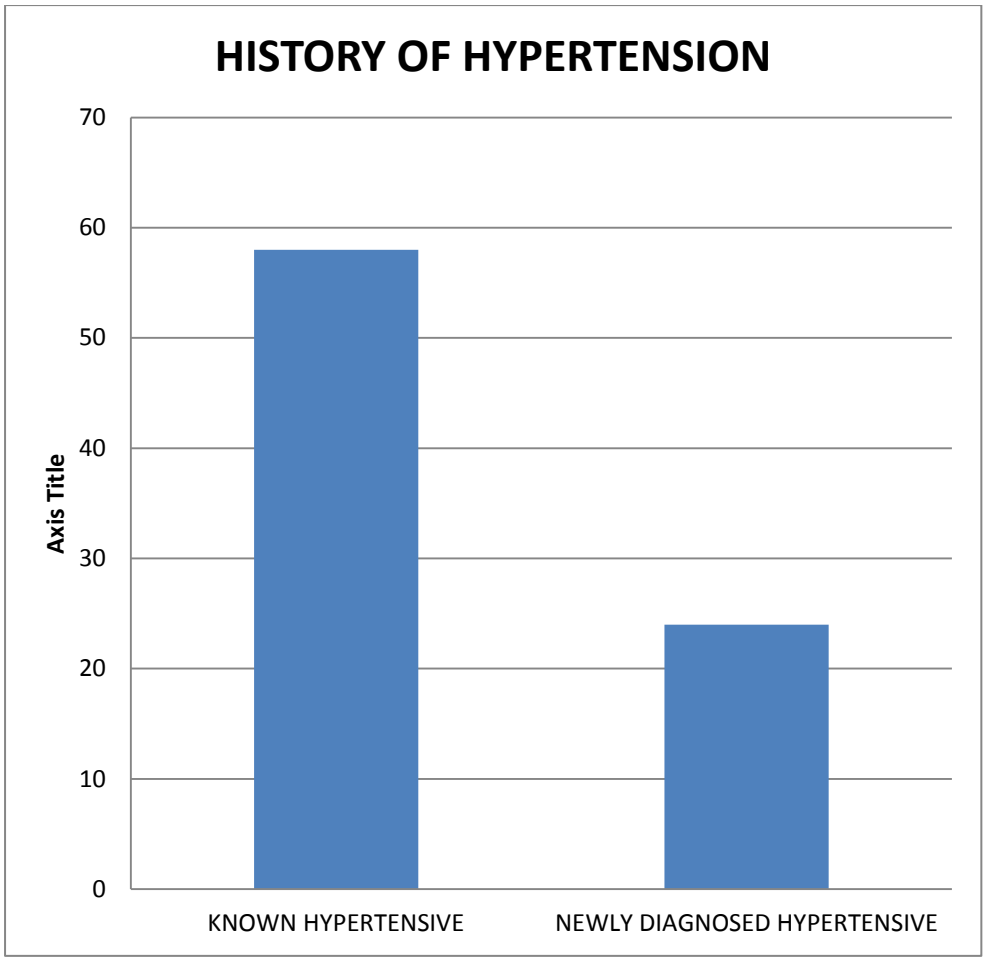
Table 3 History of study participants with hypertensive emergencies. (N=82)

S.No.	Past History	N(%)
1.	Systemic hypertension	58(70)
2.	Diabetes	34(41)
3.	Smoking	17(20)
4.	Alcohol	23(28)

In this study, patients who were presenting as hypertensive emergencies 41 % (34) were diabetic and taking either oral hypoglycemic agents or insulin.

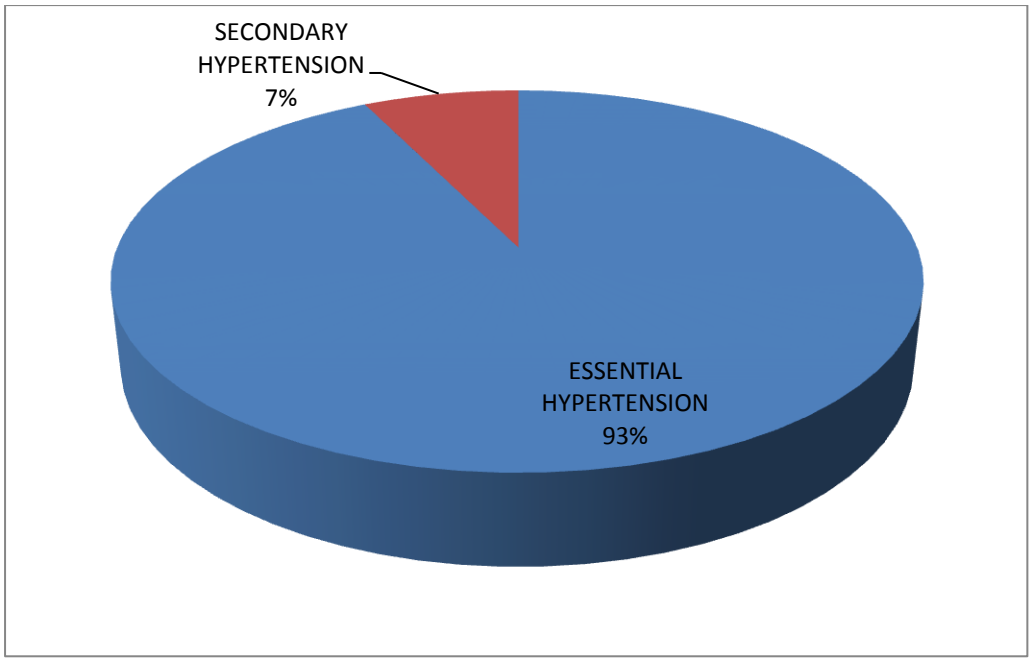
There were no smoking and alcohol history in female patients, only in male there were smoking and alcohol history. Smoking history was present in 20 % and alcohol history present in 28%.

Figure 4. History of hypertension among the patients presenting as hypertensive emergencies



In our study group among the 82 participants 70 % (58) were previously diagnosed as having systemic hypertension and were taking antihypertensive drugs and the 30 % (24) were newly diagnosed as having hypertension for the first time.

Figure 5. Cause for hypertension among the patients presenting as hypertensive emergencies



Among the 82 patients diagnosed as having systemic hypertension 93 % were having essential hypertension and the remaining 7 % were due to secondary hypertension mainly due to renal failure.

Table 4. Duration of history of Diabetes and Hypertension.

S.No.	Past History	Mean duration (±SD)
1.	Systemic hypertension	4.4(0.5)
2.	Diabetes	2.8(0.4)

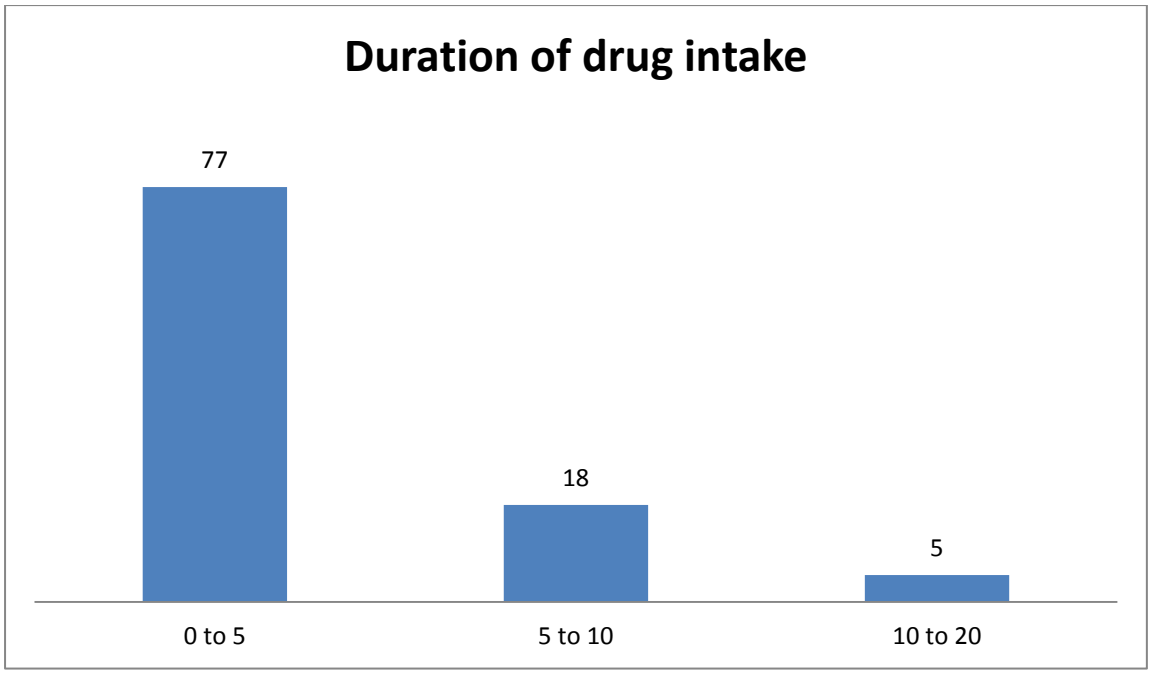
Minimum and maximum duration of hypertension is 1 and 14 years. The average duration of hypertension is 4.4 years and the standard deviation is 0.5

Minimum and maximum duration of diabetes is 1 and 20 years. The average duration of diabetes is 2.8 years and the standard deviation is 0.4

Table 5. Pattern of Medication intake for Hypertension. (N=58)

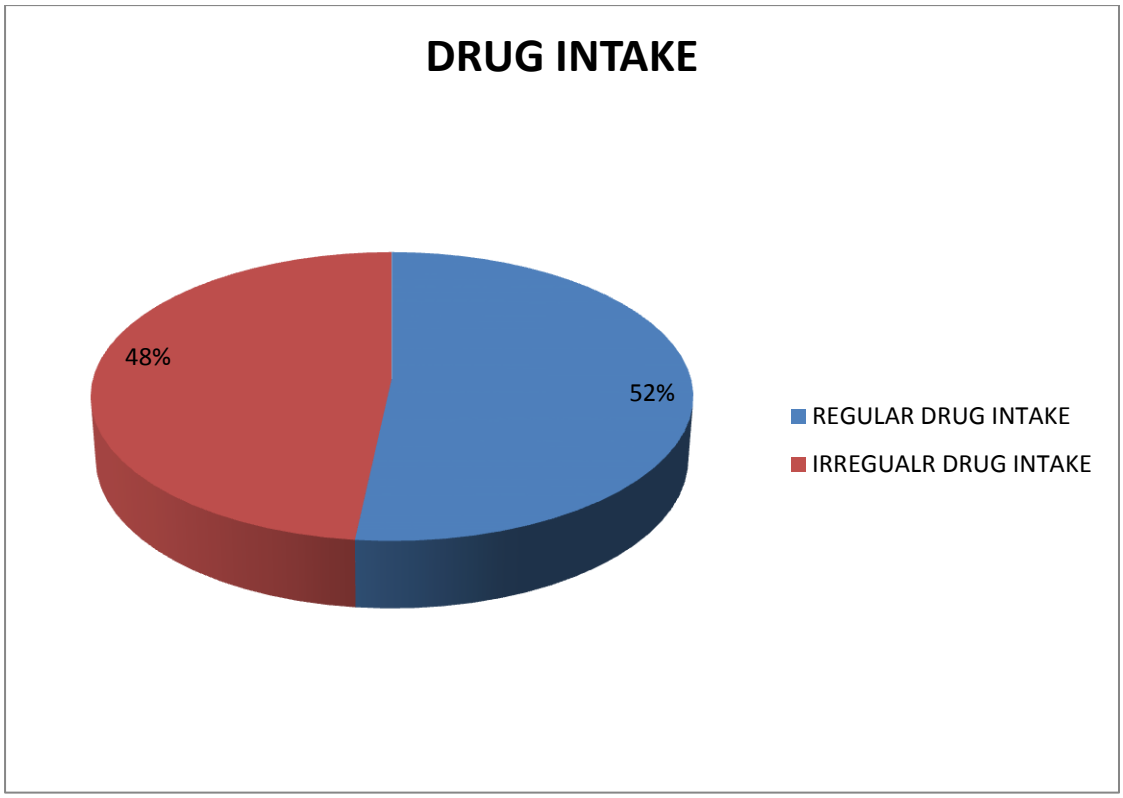
S.No.	Pattern of Medication intake	N(%)
1.	Duration of drug intake (in years)	
	0-5	45(77)
	5-10	10(18)
	10-20	3(5)
2.	Compliance in intake of medication	30(52)
	Regular	28(48)
	Irregular	

Figure 6. Duration of anti-hypertensive drugs intake among the patients presenting as hypertensive emergencies



Among the 58 patients 77 % were taking drugs for less than 5 years, 18 % were taking drugs between 5 to 10 years and 5 % were taking drugs for more than 10 years.

Figure 7. Compliance in intake of medication among the hypertensive patients



In our study group 52 % patients were regularly taking the drugs and were on regular follow up with the physician.

The 48 % were taking the drugs irregularly and not in the follow up regularly. Some of these patients were discontinued treatment after diagnosed having hypertension.

Table 6. General Examination findings of study participants with hypertensive emergencies (N=82)

S.No.	General Examination	N(%)
1.	Pallor	34(41)
2.	Pedal Edema	11(13)
3.	Elevated JVP	10(12)

In the study participants, in general examination 41 % were pallor clinically, pedal edema was present in 13 % and elevated JVP present in 12 %. Icterus, cyanosis and clubbing is not present in any of the study participants.

Table 7. Blood pressure values of study participants with hypertensive emergencies (N=82)

S.No.	Blood Pressure (mmHg)	Mean (±SD)
1.	Systolic BP	202(20)
2.	Diastolic BP	127(12)

Minimum and Maximum Systolic BP was 180 and 260 mmHg

The mean systolic BP was 202 mmHg and the SD is 20 mmHg

Minimum and Maximum Diastolic BP was 110 and 160 mmHg

The mean diastolic BP was 127 mmHg and the standard deviation is 12 mmHg

Table 8. Fundus examination of study participants with hypertensive emergencies (N=82)

S.No.	Stages of hypertensive retinopathy	N(%)
1.	Stage I	10 (12)
2.	Stage II	27(32)
3.	Stage III	9(10)
4.	Stage IV	7(8)
5.	No Changes	35(42)

Figure 8. Stages of hypertensive retinopathy among study participants with hypertensive emergencies (N=82)

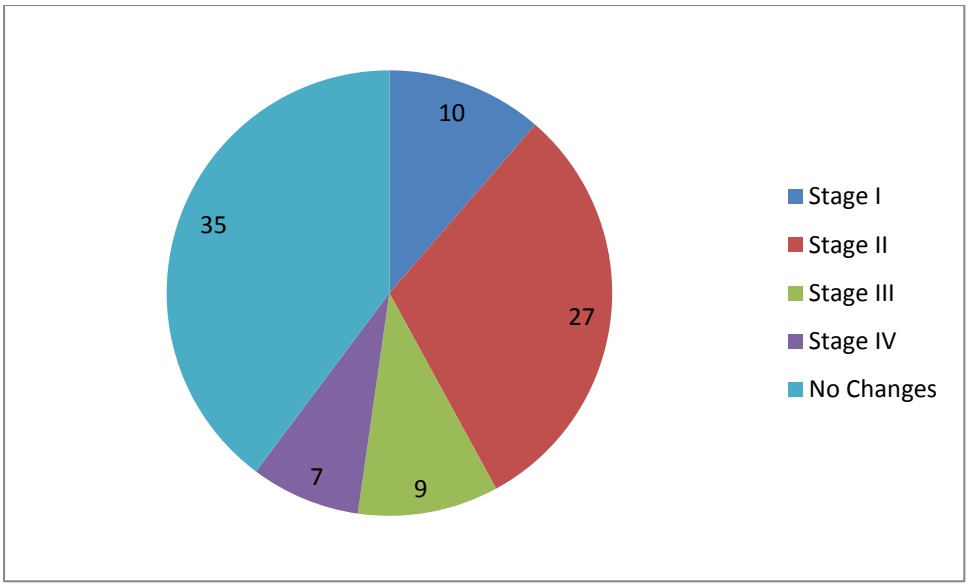


Table 9. Urine Examination findings of study participants with hypertensive emergencies (N=82)

S.No.	Urine Examination findings	N(%)
1.	Protein	20(24)
2.	Pus Cells	10(12)
3.	Sugar	9(10)
4.	Blood	2(2.4)

In study participants, proteinuria of 2+ was present in 20 patients, 10 patients were positive for pus cells in urine.

By dipstick method sugar is present in 9 patients, all of them diabetic and microscopic haematuria was present in 2 patients.

Table 10. Number of antihypertensive drugs taken by study participants.

S.No.	Number of drugs	N(%)
1.	1 Drug	8(14)
2.	2 Drugs	21(36)
3	3 Drugs	25(43)
4	4 Drugs	4(7)

Figure 9. Number of antihypertensive drugs taken by study participants

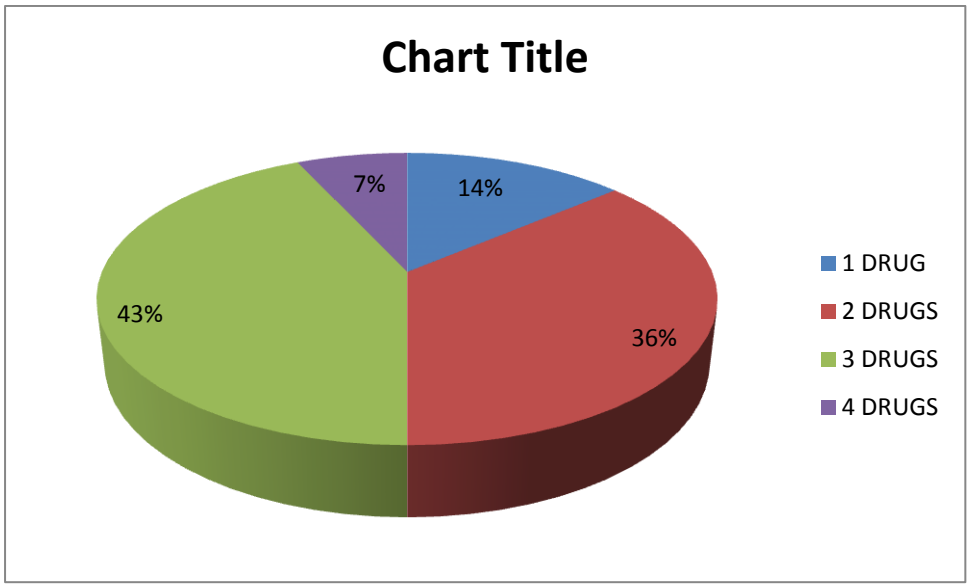


Table 11. Lab Investigation findings of study participants with hypertensive emergencies (N=82)

S.No.	Lab Investigation	Mean (±SD)
1.	Total Count (per cu.mm)	7996(2554)
2.	Hemoglobin (g/dl)	10(1.8)

The total count varied from 3800 to 13700 and the mean was 7996 with the standard deviation of 2554

The haemoglobin values ranged from 6.3 to 14 g/dl. The mean haemoglobin was 10 with standard deviation of 1.8.

Table 12. Blood parameters findings of study participants with hypertensive emergencies (N=82).

S.No.	Lab Investigation	Mean (±SD)
1.	Serum Sodium (mEq)	133(16.9)
2.	Serum Potassium (mEq)	3.7(0.81)
3.	Blood Urea (mg/dl)	42(26.0)
4.	Creatinine (mg/dl)	1.6(0.6)

Maximum and minimum value of serum sodium was 121 and 154. The mean was 133 with standard deviation of 16.9.

Maximum and minimum value of serum potassium was 2.0 and 6.1. The mean was 3.7 with standard deviation of 0.81.

Maximum and minimum value of serum urea was 12 and 132. The mean was 42 with standard deviation of 26.0.

Maximum and minimum value of serum creatinine was 0.4 and 7.8. The mean was 1.6 with standard deviation of 0.6.

Table 13. ECG findings among the study participants.

S.No.	ECG findings	N(%)
1.	LVH and Ischemic changes	43 (52)
2.	NO changes	39(48)

Figure 10. ECG findings among the study participants.

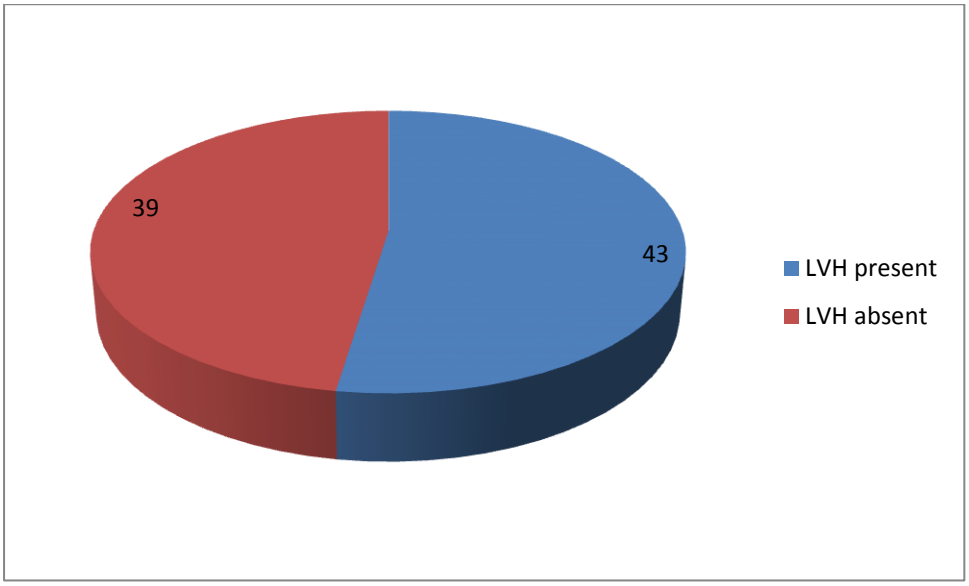


Table 14. CT Brain findings of study participants with hypertensive emergencies (N=82)

S.No.	CT findings	N (%)
1.	Intra cerebral hemorrhage	19 (23)
2.	cerebral infarct	11(13)
3.	Sub arachnoid hemorrhage	1(1.2)

The study participants who presented with any neurological signs and symptoms were subjected to CT brain imaging. In our study intracerebral haemorrhage was present in 19 patients, cerebral infarct in 11 patients and subarachnoid haemorrhage in one patient.

Table 15. Ultrasound findings of renal involvement of study participants with hypertensive emergencies (N=82)

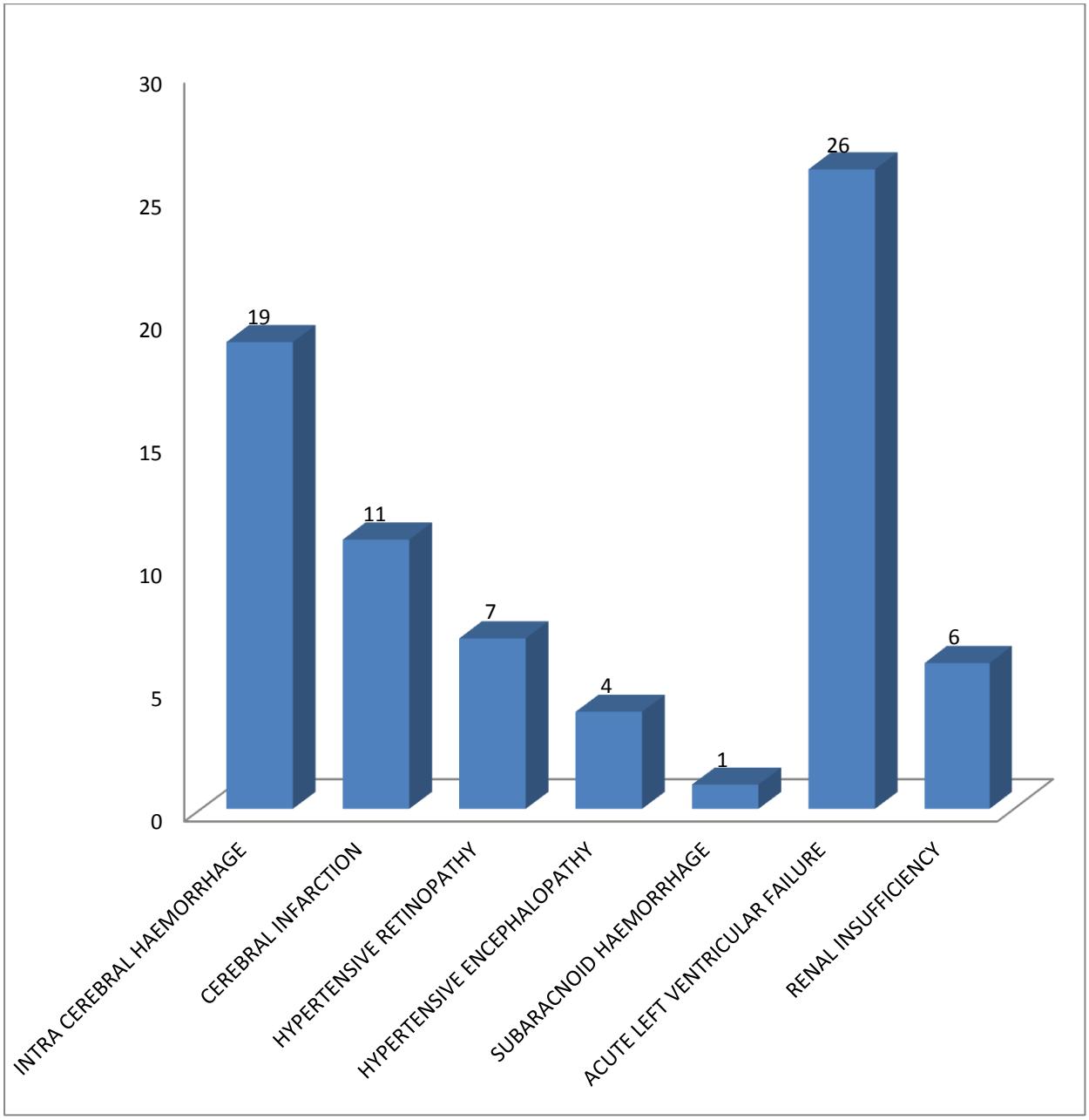
S.No.	USG findings	N(%)
1.	Medical Renal disease present	6(7)
2.	Medical Renal disease absent	73(93)

In study participants, who underwent ultrasound of abdomen it is found that 6 patients are having medical renal disease and stage 5 CKD.

Table 13. System wise presentation of Hypertensive emergency presentation.

S.No.	Presentation	N(%)
1.	Hypertensive encephalopathy	4(4.8)
2.	Cerebral infarction	11(13.4)
3.	Intracerebral haemorrhage	19(23.1)
4.	Subarachnoid haemorrhage	1(1.2)
5.	Acute left ventricular failure	26(31.7)
6.	Hypertensive retinopathy	7(8.5)
7.	Renal insufficiency	6(7.3)

Figure 11. System wise presentation of Hypertensive emergency.



Discussion

Zampaglione B et al (16) did study in Italy to assess the prevalence of hypertensive emergencies and target organ damage during 24 hours. In this study

hypertensive crises (76% urgencies, 24% emergencies) represented more than one fourth of all medical urgencies-emergencies.

The most frequent signs of presentation were headache (22%), epistaxis (17%), faintness, and psychomotor agitation (10%) in hypertensive urgencies and chest pain (27%), dyspnea (22%), and neurological deficit (21%) in hypertensive emergencies.

Types of end-organ damage associated with hypertensive emergencies included cerebral infarction (24%), acute pulmonary edema (23%), and hypertensive encephalopathy (16%) as well as cerebral hemorrhage,

Hypertension that was unknown at presentation was present in 8% of hypertensive emergencies and 28% of hypertensive urgencies.

Luis Alfonso et al (18), did a study to evaluate the clinical significance of troponin T elevation in hypertensive emergencies

Hypertensive emergencies (HEs) are frequently accompanied with the release of cardiac troponin I (cTnI). A retrospective analysis was performed on patients (n=567) with a diagnosis of HE admitted to two tertiary care centers

Predictors of cTnI were age, history of hypercholesterolemia, blood urea nitrogen level, pulmonary edema, and requirement for mechanical ventilation.

During a mean follow-up period of 3.1 years, there were 211 deaths (37%).

He found that cTnI elevation commonly occurs in the setting of HEs. Despite a high incidence of adverse clinical outcomes, cTnI elevation was not an independent predictor of mortality in this population

Rashed A et al (10), in this study to assess the prevalence and presentation of hypertensive emergencies he found that LVF- 38%, ACS – 33%, Stroke – 29% were common presentation. Chest pain and headache are most common symptoms.

Ellenga MB et al (19), did a study in Congo to determine the prevalence and clinical characteristics of hypertensive emergencies.

The disease underlying the hypertensive emergency was stroke with 38 cases (50%), heart failure in 20 (26.3%), hypertensive encephalopathy in 11 (14.4%), malignant hypertension in 9 (11.8%), and renal failure in 10 (13.1%).

The mean length of emergency treatment was 14.7 hours (range, 5 to 48 hours). Eight deaths (10.5%) occurred during hospitalization in the emergency department.

José Fernando Vilela Martin et al (13). found that hypertensive crisis, accounted for 0.5% of all clinicosurgical emergencies, of which, 273 (60.4%) were hypertensive urgencies and 179 (39.6%) were hypertensive emergencies.

Eighteen percent of the patients ignored their hypertensive condition. Smoking and diabetes were risk factors associated with the development of a hypertensive crisis in 1/4 and 1/5 of the patients, respectively.

Ischemic stroke and acute pulmonary edema were the most common hypertensive emergencies, being in accordance with the most frequently found clinical manifestations of neurologic deficit and dyspnea.

Francisco das Chagas Monteiro Júnior et al (14). did a study to assess the prevalence of True Hypertensive Crises and Appropriateness of the Medical

Management in Patients with High Blood Pressure in a General Emergency Room.

He found that Criteria for the characterization of a hypertensive crisis were present in only 27 patients (16%), and all were classified as urgencies. Medical management was considered appropriate in 72 cases (42.6%), and was neither influenced by specialty nor by the physician's experience.

Blood pressure levels, but not the presence or absence of symptoms, were predictive of the use of antihypertensive medication.

Seth R et al (11), found that, the mean age was 54.3±15.6 years; 64% were female; 46% were black; 90% had diagnosed hypertension. The mean presenting BP was 198±27.6/109±17.3 mm Hg; 66% had systolic BP >180 mm Hg, and 38% had diastolic BP >110 mm Hg.

Initially, 30% were not on antihypertensives, and 28% were on monotherapy. Headache (42%) and dizziness (30%) were most frequently reported symptoms.

Hector Gonzalez Pacheco et al (20), found that of all of the patients admitted to a coronary care unit, 538 experienced a hypertensive crisis, which represented 5.08% of all admissions.

Hypertensive emergency was predominant in 76.6% of the cases, which corresponded to acute coronary syndrome and acute decompensated heart failure in 59.5% and 25.2% of the cases, respectively.

A pattern of predominant systolic hypertension (_180/_119 mm Hg) was most commonly observed in the hypertensive crisis group (71.4%) and the hypertensive emergency group (72.1%). The medications that were most commonly used at onset included intravenous vasodilators (nitroglycerin in 63.4% and sodium nitroprusside in 16.4% of the patients).

The overall mortality rate was 3.7%. The mortality rate was 4.6% for hypertensive emergency cases and 0.8% for hypertensive urgencies cases.

James E Tisdale (12) did a study to identify independent risk factors for development of hypertensive crisis. He found that the average blood pressure during emergency Department presentation in patients with hypertensive crisis was 197 ± 21/108 ± 14 mmHg.

Less successful out-patient systolic blood pressure control was an independent risk factor for hypertensive crisis. Higher out-patient diastolic blood pressures and history of heart failure trended towards independence as risk factors.

Sanjay k. Gandhi et al (15), found that in patients with hypertensive pulmonary edema, a normal ejection fraction after treatment suggests that the edema was due to the exacerbation of diastolic dysfunction by hypertension — not to transient systolic dysfunction or mitral regurgitation.

Sharma BK et al (17), found that among the 135 patients admitted with malignant hypertension. The etiology was essential hypertension in 88 patients and a secondary cause in 47 patients.

Secondary causes included a renovascular etiology in 20 patients, renal parenchymal disease in 19, pheochromocytoma in 6 and Conn's syndrome and adrenal carcinoma in one patient each.

Among the 20 patients with renovascular hypertension, Takayasu's arteritis was seen in 15 (75%). The mean age of patients with essential hypertension was 41.7 + 1.14 years, the mean age in patients with secondary hypertension was 33.2 + 1.96 years.

No	Author	Study setting	Conclusions
1	Hector Gonzalez Pacheco et al (9).	2011 Mexico city	Hypertensive crisis in coronary care unit represented 5.08% of all admissions. Hypertensive emergency was predominant in 76.6% of the cases, which corresponded to acute coronary syndrome and acute decompensated heart failure in 59.5% and 25.2% of the cases, respectively
2	Rashed et al (10).	2010 Saudi	LVF- 38%, ACS – 33%, Stroke – 29% Chest pain and headache are most common presentation.
3	Seth R et al (11).	2005 New York	The mean presenting BP was 198±27.6/109±17.3 mm Hg; 66% had systolic BP >180 mm Hg, and 38% had diastolic BP >110 mm Hg. Initially, 30% were not on antihypertensives, and 28% were on monotherapy. Headache (42%) and dizziness (30%) were most frequently reported

			symptoms. Presentation was most often attributed to running out of medication(16%)
4	Tisdale JE et al (12).	2004 USA	The average blood pressure during Emergency Department presentation in patients with hypertensive crisis was 197 ± 21/108 ± 14 mmHg. Less successful out-patient systolic blood pressure control was an independent risk factor for hypertensive crisis
5	José Fernando Vilela Martin et al(13)	2003 Brazil	Hypertensive crises accounted for 0.5% of all emergency cases studied and for 1.7% of all clinical emergencies, hypertensive urgency being more common than hypertensive emergency. Ischemic stroke and acute pulmonary edema were the most frequent target-organ lesions.
6	Francisco das Chagas Monteiro Júnior et al(14)	2003 Brazil	In the population analyzed, less than one fifth of the patients seen in an emergency room with a presumed hypertensive crisis met defined criteria for this diagnosis. Medical management was considered appropriate in less than half of the occurrences

7	Sanjay K. Gandhi et al(15)	2000 USA	In patients with hypertensive pulmonary edema, a normal ejection fraction after treatment suggests that the edema was due to the exacerbation of diastolic dysfunction by hypertension — not to transient systolic dysfunction or mitral regurgitation
8	Zampaglione B et al(16)	1993, Italy	Types of end-organ damage associated with hypertensive emergencies included cerebral infarction (24%), acute pulmonary edema (23%), and hypertensive encephalopathy (16%) as well as cerebral hemorrhage, which accounted for only 4.5%.
9	Steven Shea et al(17)	1992 Columbia	Characteristics of both the health care system and patients behaviour are associated with severe uncontrolled hypertension.

Conclusion

The prevalence of hypertensive emergencies in our setup is 0.68

In this 42 % were more than 60 years. Headache and giddiness were the most common presenting symptonm

The hypertension was undiagnosed in nearly one third of patients

In hypertension 93 % were essential hypertension and 7% is due to renal failure

Nearly 50% of hypertensive emergencies were on irregular treatment and follow up. Majority were recently diagnosed

The most common system wise presentation is neurological. The most common is cardiovascular system.

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Prevalence and clinical characteristics of hypertensive emergencies in tertiary care teaching institute , Chennai

Patient ID No: : Date:

1. Name : Age/sex

2. Address :

Presenting complaints	**Yes/No**	**Duration**
Headache		
Altered mental status		
Seizure		
Neurological deficit		
Visual changes		
Nausea/Vomiting		
Breathlessness		
Chest pain		
Giddiness		
Epistaxis		
Decreased urine output		
Haematuria		

Other complaints		

Past history

SHT		DM		CAD	
CVA		Renal disease		other	

Family history

SHT		DM		CAD	
CVA		RENAL disease		other	

Personal history

Smoker		Duration		Amount	
Alcoholic		Duration		Amount	

Drug history

Drugs intake/years	
No of drugs	
Details of drug	
Compliance with drugs	
Last known BP	
Adequate control in the past	
Previous crisis	
Recent change of drugs	
Dosing of drugs	
Reason for discontinuing drugs	

General examination

Anaemia		Jaundice		Cyanosis	
Clubbing		Pedal edema		JVP	

Vitals

Systolic BP		MAP	
Diastolic BP		HR	

Systemic examination

Cardiovascular	
Respiratory	
Abdomen	
CNS	
Fundus	

Investigations

Urine R/E	
Blood urea/creatinine	
Serum electrolytes	
Complete blood count	
ECG	
X Ray chest	
Echo	
Lipid profile	
USG abdomen	
Cardiac enzymes	
Peripheral smear	
CT brain	

Annexure 2: Informed consent

Govt. Stanley medical college, chennai – 600001

Informed consent

Prevalence and clinical characteristics of hypertensive emergencies in Tertiary care centre in chennai.

Place of study: govt. Stanley medical college, Chennai

I ……. have been informed about the details of the study in my own language.

I have completely understood the details of the study.

I am aware of the possible risks and benefits, while taking part in the study.

I understand that I can withdraw from the study at any point of time and even then, I can receive the medical treatment as usual.

I understand that I will not get any money for taking part in the study.

I will not object if the results of this study are getting published in any medical journal, provided my personal identity is not revealed.

I know what I am supposed to do by taking part in this study and I assure that I would extend my full cooperation for this study.

Volunteer:	Witness:
Name and address	Name and address
Signature/thumb impression:	Signature/thumb impression
Date:	Date:

Investigator

Signature and date

அரசு. ஸ்டான்லி மருத்துவ கல்லூரி, சென்னை – 600001

Prevalence and clinical characteristics of hypertensive emergencies in Tertiary care centre in chennai.

நான் இந்த ஆராய்ச்சியில் விவரங்களை முற்றிலும் புரிந்து கொண்டேன்.

ஆய்வில் பங்கு எடுத்து போது, சாத்தியமான அபாயங்கள் மற்றும் பயன்களை பற்றி நான் அறிந்துள்ளேன்.

நான் எந்தவொரு வேளையிலும் ஆய்வில் இருந்து திரும்ப முடியும், அதன் பின்னர், நான் வழக்கம் போல் மருத்துவ சிகிச்சை பெற முடியும் என்று புரிந்துகொள்கிறேன்

நான் ஆய்வில் பங்கு எடுத்து பணம் எதையும் பெற முடியாது என்று அறிந்துள்ளேன்.

இந்த ஆய்வின் முடிவுகள் எந்த மெடிக்கல் ஜர்னலில் வெளியிடப்பட இருந்தால் நான் எதிர்க்கவில்லை, என் தனிப்பட்ட அடையாளத்தை வெளிப்படுத்தப்பட்டு இருக்க கூடாது.

நான் இந்த ஆய்வில் பங்கெடுப்பதன் மூலம் நான் என்ன செய்ய போகிறேன் என்று தெரியும்

நான் இந்த ஆய்வில் என் முழு ஒத்துழைப்பையும் கொடுப்பேன் என்று உறுதியளிக்கிறேன்.

தன்னார்வளர்	சாட்சி
பெயர் மற்றும் முகவரி	பெயர் மற்றும் முகவரி
கையொப்பம் / விரல் ரேகை:	கையொப்பம் / விரல் ரேகை:

ஆராய்ச்சியாளராக
கையொப்பம் மற்றும் தேதி

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Prevalence and clinical characteristics of hypertensive emergencies in tertiary care centre in Chennai.

Principal Investigator : Dr. Karthikeyan B

Designation : PG in MD (General Medicine)

Department : Department of General Medicine
Government Stanley Medical College,
Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 01.04.2014 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

INSTITUTIONAL ETHICAL COMMITTEE,